FINAL DRAFT

BASELINE RISK ASSESSMENT

FOR THE SCIENTIFIC CHEMICAL PROCESSING (SCP), INC. SITE

Prepared for:

U.S. Environmental Protection Agency
Region II
26 Federal Plaza
New York, New York 10278

Clement Associates Incorporated

Environmental and Health Science

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The SCP site is currently the subject of a Comprehensive Environmental
Response, Compensation and Liability Act (CERCLA, as amended), Remedial
Investigation/Feasibility Study (RI/FS). The site is classified as an
enforcement lead site for which potentially responsible parties (PRPs) are
conducting the RI/FS. At the request of Region II of the U.S. Environmental
Protection Agency (USEPA) this Baseline Risk Assessment (BRA) was prepared
under the REM III Superfund contract. It is a stand-alone document, conducted
independently of the PRP's RI and RA efforts, but which relies on the Remedial
Investigation conducted by Dames and Moore (1990) for the PRPs as a primary
source of information.

The SCP site is located in Carlstadt Township, Bergen County, New Jersey.
Solvent refining, solvent recovery, recycling industrial wastes and other
waste storage, treatment and disposal activities occurred at the SCP site from
the early 1960s until at least 1980. The site received or stored a wide
variety of industrial wastes, including solvents, PCBs and numerous other
hazardous substances.

The SCP site is located in a commercial/industrial area zoned for light
industry. Hotel and restaurant uses are also permitted on the site. The site
is bounded by Peach Island Creek on the northeast, Paterson Plank Road on the
southwest, Gotham Parkway on the northwest, and an industrial facility on the
southeast. The site is underlain by three water-bearing units: (1) a shallow
water table aquifer; (2) a glacial till aquifer (overlain by a clay/silt unit

above); and (3) the bedrock aquifer which underlies the till aquifer, and which is utilized as a public drinking water supply. The water table aquifer is located approximately one to two feet beneath the surface of the site. This aquifer and the soils above the clay unit are the most highly contaminated media at the site. A broad spectrum of organic and inorganic hazardous substances exist in this zone, including PCBs, volatile organic compounds (VOCs), pesticides, and metals, many of which are known or suspected human carcinogens. Numerous hazardous substances have migrated from this zone into the underlying till aquifer, and into Peach Island Creek. Lateral flow in the water table aquifer appears to move radially across all borders of the site (with some flow towards the site's interior). In addition, there is vertical flow downward into the till aquifer. Flow in the till aquifer appears to move in a northwesterly direction (i.e., towards Gotham Parkway). In addition, the till aquifer is subject to tidal influences.

E.1 SELECTION OF CHEMICALS FOR EVALUATION

Forty-three chemicals of concern which exist in the soil and/or ground water at the site (see Table E-1) were selected for detailed evaluation in this Baseline Risk Assessment. The sampling data used in this selection process and in the evaluation were collected and analyzed as part of the Dames and Moore (1990) RI in addition to two samples collected from the bedrock aquifer in 1989 and analyzed by USEPA during continuation of the Dames and Moore RI work. The Dames and Moore (1990) RI data were obtained from the raw laboratory data reports (ETC December 1987) provided to USEPA by Dames and Moore.

TABLE E-1

SUMMARY OF SELECTED CHEMICALS OF CONCERN AT THE SCP SITE

| | MEDIUM | | | |
CHEMICAL[a]	SOIL	GROUND WATER	SURFACE WATER	SEDIMENTS
Volatile Organic Compounds				
benzene	X	X	ND	X
chlorobenzene	X	X	X	X
chloroform	X	X	X	X
1,1-dichloroethane	X	X	X	ND
1,2-dichloroethane	X	X	X	X
1,1-dichloroethylene	X	X	ND	ND
1,2-trans-dichloroethylene	X	X	X	X
ethylbenzene	X	X	ND	X
methyl ethyl ketone	X	X	X	X
methlyene chloride	X	X	X	X
1,1,2,2-tetrachloroethane	X	X	ND	ND
tetrachloroethylene	X	X	ND	X
toluene	X	X	X	X
1,1,1-trichloroethane	X	X	X	X
trichlorethylene	X	X	X	X
vinyl chloride	X	X	ND	ND
xylenes (o+p,m)	X	X	X	X
Pesticides/PCBs				
aldrin	X	ND	ND	ND
dieldrin	X	ND	ND	X
polychlorinated biphenyls (PCBs)	X	X	ND	X
Semi-Volatile Compounds				
bis-2(ethylhexyl)phthalate	X	X	ND	X
butyl benzyl phthalate	X	X	ND	X
2-chloronaphthalene	X	X	ND	X
1,2-dichlorobenzene	X	X	ND	X
2,4-dimethylphenol	X	X	ND	X
di-n-butyl phthalate	X	X	ND	X
di-n-octyl phthalate	X	X	ND	X
isophorone	X	X	ND	ND
nitrobenzene	X	X	ND	ND
PAHs, carcinogenic	X	X	ND	X
PAHs, noncarcinogenic	X	X	ND	X
phenol	X	X	ND	X

TABLE E-1 (Continued)

SUMMARY OF SELECTED CHEMICALS OF CONCERN AT THE SCP SITE

| | MEDIUM | | | |
CHEMICAL[a]	SOIL	GROUND WATER	SURFACE WATER	SEDIMENTS
Inorganic				
antimony	X	ND	ND	ND
arsenic	X	X	ND	X
cadmium	X	X	ND	X
chromium	X	X	X	X
cyanide	X	X	ND	X
lead	X	X	ND	X
mercury	X	X	X	X
nickel	X	X	X	X
selenium	X	ND	ND	--
silver	X	ND	ND	X
zinc	X	X	X	X

[a] Only chemicals selected in one or more medium are included.

PAH = Polycyclic aromatic hydrocarbon.
ND = Not detected.
-- = Not selected (see text for discussion).

The selected chemicals fall within the following classes of contaminants: VOCs (17), pesticides (2), PCBs, semi-volatile compounds (12) and inorganic chemicals (11).

The highest concentrations for almost all the chemicals of concern listed in Table E-1 are found in the soil and/or ground water located above the clay lens at the site. The levels of contaminants in this zone generally exceed those found anywhere else at or near the site (i.e., below the clay, in the Creek, or in ground water adjoining the site).

In selecting chemicals from among those detected at and near the site, factors that were considered included frequency of detection, concentrations detected, detection in more than one environmental medium, mobility potential, effects of the chemicals, and, for inorganics, presence in soils at levels above background levels. Table E-1 summarizes both the final list of chemicals of concern and the media in which they were detected at the site.

For each of these chemicals, health criteria (i.e., quantitative dose-response values) for both carcinogenic and noncarcinogenic effects associated with exposure were collected. The primary source of this information was the U.S. Environmental Protection Agency's (USEPA's) Integrated Risk Information System (IRIS) and Health Effects Assessment Summary Tables (HEASTs). The health criteria for carcinogenic effects are slope factors developed by USEPA's Carcinogen Assessment Group (CAG). The health criteria for noncarcinogenic effects are reference doses (RfDs) generally developed by USEPA's RfD Work Group.

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The following statements and conclusions can be made regarding the selected chemicals of concern:

(1) All are hazardous substances under CERCLA.

(2) None of the VOCs, pesticides, PCBs and semi-volatile compounds listed originate from natural sources but yet many of these chemicals exist at grossly elevated levels at the site.

(3) Some are possible human carcinogens (e.g., butyl benzyl phthalate, 1,1-dichloroethylene, isophorone, 1,1,2,2-tetrachloroethane).

(4) Many of the selected chemicals are probable human carcinogens (e.g., PCBs, chloroform, 1,2-dichloroethane, methylene chloride, trichloroethylene, cadmium [inhalation only]).

(5) Some are known carcinogens in humans (e.g. vinyl chloride, arsenic, benzene).

(6) Many exist in the water table aquifer at the site at levels which far exceed (often by orders of magnitude) the Maximum Contaminant Levels (MCLs) established for such substances pursuant to the Federal Safe Drinking Water Act (e.g., benzene and vinyl chloride [known human carcinogens], and chlorobenzene, chloroform, 1,2-dichloroethane, 1,2-trans-dichloroethylene, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane).

(7) Many exist in the till aquifer at the site at levels which exceed (often by orders of magnitude) the MCLs which were established for substances pursuant to the Federal Safe Drinking Water Act (e.g., 1,2-dichloroethane, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane, chlorobenzene, and vinyl chloride [a known human carcinogen]).

(8) Some chemicals exist in the bedrock aquifer at levels which exceed (in some cases by orders of magnitude) MCLs (e.g., 1,2-dichloroethane, trichloroethylene).

(9) Some chemicals exist in the shallow water table aquifer at the site at levels which far exceed the Class GW-2 drinking water standards set by the State of New Jersey for such chemicals in this aquifer (e.g., PCBs).

(10) Many of the selected chemicals detected at the site are known to cause acute and/or chronic health effects (other than carcinogenic) in humans if ingested, inhaled, or dermally contacted in sufficient quantities.

(11) Many of the selected chemicals which exist at the site and which were also detected in the sediment of Peach Island Creek are known to be acutely and/or chronically toxic to aquatic organisms.

(12) Some of the selected chemicals which exist at the site and which were also detected in the sediment of Peach Island Creek are known to bioaccumulate and biomagnify in certain aquatic species (e.g., PCBs).

(13) Many of the selected chemicals are highly mobile in ground water (as indicated in Table 3 of the Dames and Moore [1990] RI).

(14) Almost all of the selected VOCs (14 of 17) which exist in the soil and ground water at the site were also detected in either the water column and/or sediment in Peach Island Creek.

(15) Almost all of the selected semi-volatile compounds (10 of 12) which exist in the soil and ground water at the site were also detected in the sediment in Peach Island Creek.

(16) Some of the selected chemicals which exist at the site were also detected in Peach Island Creek at levels which exceed the applicable standards for that creek (e.g., copper, mercury, nickel, zinc).

(17) The site is presently uncapped and open to the atmosphere. Many of the chemicals discovered at the site are known to be capable of volatilizing into the atmosphere and thereby migrating away from the site in ambient air.

(18) The site receives approximately seven million gallons per year of precipitation, some of which flows off the site in the form of surface runoff into Peach Island Creek. Some precipitation will also infiltrate into the shallow water table aquifer. No controls or catchment structures exist to prevent this migration at present. Therefore, many of the hazardous substances listed in Table E-1 may migrate into this creek, especially during and shortly after storm events with consequential unknown impacts on aquatic biota.

E.2 HUMAN EXPOSURE ASSESSMENT

Potential pathways by which human populations (workers, residents, etc.) could be exposed to chemicals at or originating from the site under current land use or hypothetical future land use conditions were identified and selected for evaluation. An important first step in identifying exposure pathways is to

consider the mechanisms by which the chemicals of concern at the site may migrate in the environment.

E.2.1 POTENTIAL MIGRATION OF SITE-RELATED CONTAMINANTS

The potential migration routes for chemicals at the SCP site include:

(1) Migration from the soils into the ground water at the site;

(2) Migration from the shallow water table aquifer downward into the till aquifer;

(3) Migration from the till aquifer into the bedrock aquifer (which is presently used as a public water supply);

(4) Surface runoff from the site into Peach Island Creek;

(5) Migration of ground water directly into Peach Island Creek;

(6) Lateral migration of on-site ground water to off-site areas;

(7) Migration into the air by volatilization or particulate suspension; and

(8) Migration in Peach Island Creek of surface water and sediments.

An assessment of the potential environmental effects posed by some of these routes of migration was not possible given the limited data available at this time (e.g., lateral migration into off-site ground water areas). Analysis of samples collected from soil, ground water, surface water, and sediment at the SCP site indicate, however, that chemicals of concern together with their transformation products are present in several or all of these media.

Many of the compounds detected in soil at the site will migrate due to the presence of infiltrating precipitation and the downward hydraulic gradient

between the water table and till aquifers (Dames and Moore 1990). The substituted simple aromatics (e.g., chlorobenzene), chlorinated aliphatics (e.g., tetrachloroethylene) and phenols are expected to be more mobile than the other chemicals of concern in soil. The propensity of these chemicals to be mobile in soils is observed at the SCP site, where the substituted simple aromatics and chlorinated aliphatics have been observed in all soil depths sampled and in the water table, till, and bedrock aquifers. Based on the presence of several substituted simple aromatics and chlorinated aliphatics at elevated levels within the clay and in the underlying till aquifer, and at detectable levels in the bedrock aquifer, it can be concluded that hazardous substances are migrating into the bedrock aquifer.

The chlorinated pesticides, PAHs, PCBs, and phthalate esters detected in soils are generally not as mobile in soil as the chemicals mentioned above. Many of these chemicals were, however, present at all the soil depths sampled at the SCP site. This is particularly true for PCBs and many of the PAHs indicating that downward transport is still occurring. PCBs were present in unfiltered samples collected from both the water table and till aquifers, and in filtered water table aquifer samples at levels exceeding their solubility. This may result from the presence of organic solvents which may enhance the solubility (and thus mobility) of PCBs in ground water and in soil.

Many of the chlorinated aliphatics and substituted simple aromatics detected in Peach Island Creek adjacent to the site were also detected in the water table and till aquifers and in soils at the site (e.g., 1,1,1-trichloroethane, 1,2-trans-dichloroethylene). The similarity of compounds detected in Peach

Island Creek as compared to those detected in the ground water and soil at the SCP site strongly suggest that chemical contaminants have migrated via ground water discharge and/or surface runoff from the site into the creek. However, given the limited surface water sampling program undertaken and the tidal nature of the creek, it is difficult to identify definitively the extent to which site-related contaminants may be migrating into the creek from the site. There are, however, clear similarities between the types of chemicals detected in sediments and those detected on site in ground water and soil. These data further suggest that contaminants are migrating into Peach Island Creek from the site.

Chemicals present at the SCP site may migrate into the air in two ways, by volatilization or by suspension of soil (i.e., generation of fugitive dusts). Of the chemicals of concern at the SCP site, the chlorinated aliphatics (e.g., 1,1-dichloroethane, 1,2-dichloroethane, 1,1-dichloroethylene, 1,2-trans-dichloroethylene, methylene chloride, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene, and vinyl chloride) and some of the substituted simple aromatics (e.g., benzene, 1,2-dichlorobenzene, toluene, and total xylenes) tend to volatilize readily from contaminated soils and water. While the other chemicals of concern such as the pesticides, phthalate esters, PAHs, and PCBs are less volatile, they still may be emitted from site soils into the air. Fugitive dust emissions could also occur at the SCP site in areas that are unpaved or unvegetated.

Biological and chemical processes that occur in the soil can also be important in determining the ultimate fate of the organic chemicals found at the SCP

site. These processes can, for example, produce more toxic and/or more mobile breakdown products. In most cases, an organic chemical occuring in the natural environment is not broken down immediately to carbon dioxide and water by a microorganism, but is metabolized to an intermediate which is in turn further degraded. These intermediates are typically more water soluble than the parent compound and are therefore more mobile. Many of the organic intermediates are also more toxic. For example, it is possible that the vinyl chloride in the water table and till aquifers at the SCP site occurs as a result of the transformation of the unsaturated higher molecular weight chlorinated aliphatics (e.g., trichloroethylene). Vinyl chloride is stable with respect to further biological and/or chemical transformation and is likely to persist unless it has an opportunity to volatilize or leach from soil. PCBs, which are comprised of mixtures of polychlorinated biphenyl congeners, may be metabolized by microorganisms present in the environment. Metabolism of one PCB congener will sequentially yield PCB congeners of lower molecular weight and greater solubility along with other metabolic byproducts such as PCB alcohols and/or ethers. The PAHs present in soil can also be biodegraded.

Most of the hydrophobic organics (e.g., PAHs, PCBs, dieldrin) and inorganics that may enter Peach Island Creek from the site tend to adsorb to organic and inorganic particulate matter in the waterway and subsequently deposit in the sediments. This contaminant burden usually remains relatively near the source, with concentrations generally decreasing with distance from the source. Many of the chemicals of concern in sediments in Peach Island Creek (e.g., trichloroethylene, toluene, Aroclor 1242, xylenes, and ethylbenzene)

were in fact detected at their highest levels immediately adjacent to the site.

E.2.2 IDENTIFICATION OF EXPOSURE PATHWAYS

Based on a review of the site area and the results of the site investigation, a set of pathways through which humans may be exposed currently or in the future to site-related contaminants was identified for detailed evaluation. The exposure pathways that were evaluated in the risk assessment for both current and future site and nearby land use conditions were as follows:

Current Site and Nearby Land Use Conditions

- Direct Contact with Site Surface Soil by Trespassers
- Inhalation of Volatilized Organics by Nearby Residents and Workers
- Inhalation of Suspended Soil by Nearby Residents and Workers

Future Site and Nearby Land Use Conditions

- Direct Contact with Surface Soil by Future On-Site Workers
- Inhalation of Volatilized Organics by Future On-Site Workers
- Inhalation of Suspended Soil by Future On-Site Workers
- Ingestion of Ground Water by Future On-Site Workers
- Direct Contact with Subsurface Soil by Future Construction Workers

Other potential pathways of exposure which may exist but which were not evaluated in the assessment (e.g., due to insufficient sampling data) include: incidental ingestion of surface water and sediments from Peach Island Creek, ingestion of fish or shellfish from Peach Island Creek, and ingestion of any ground water in the vicinity of the site. In addition, exposures to chemicals in ground water through routes other than ingestion (e.g., inhalation of

volatiles released from water into indoor air) were not quantitatively evaluated.

In order to evaluate exposures for each selected pathway, scenarios were developed based on estimates regarding the extent, frequency, and duration of exposures. In addition, the concentrations to which individuals might be exposed were calculated based on the site sampling data except for the inhalation pathways for which emission and dispersion models were used to estimate air concentrations. These concentrations are referred to as exposure point concentrations.

For each exposure pathway, the potential exposure to individuals was estimated for both an average case and, in accordance with recent USEPA guidance (USEPA 1989a) at the request of USEPA Region II, a reasonable maximum exposure (RME) case. The average case combines average exposure point concentrations with average estimates for the extent, frequency, and duration of exposure. The average case is designed to be roughly representative of realistic exposures a "typical" individual might experience. For the same receptor locations, the RME case combines the maximum exposure point concentrations with RME values describing the extent, frequency, and duration of exposure. The RME scenario incorporates the exposure parameter values recommended in USEPA's (1989a) Superfund guidance to the extent possible given that this project was well underway at the time the new USEPA guidance was released. The RME case is designed to represent an upper bound on potential exposures; that is, predicted exposures are likely to overestimate expected risks but would not underestimate actual risks.

E.3 HUMAN RISK CHARACTERIZATION

Risks from the above exposures were evaluated first by comparing concentrations of chemicals in the contaminated exposure medium (e.g., ground water) at point of potential exposure, to State or Federal environmental standards, criteria, or guidance that were identified as "Applicable or Relevant and Appropriate Requirements" (ARARs) or other relevant guidance. In addition, exposures were also evaluated by quantitative risk assessment.

The soil sampling data from the site indicate that numerous chemicals exceed the NJDEP soil cleanup objectives as shown in Table E-2. These include total volatile organics, total base neutral and acid extractable compounds, PCBs, arsenic, chromium, cadmium, lead, mercury, and zinc.

A wide variety of chemicals also exceeded federal and state standards and guidelines for ground water. The ARARs and other guidance that were used in this comparison were federal maximum contaminant levels (MCLs) and MCL goals (MCLGs), federal ambient water quality criteria for protection of human health adjusted for drinking water exposures only, state MCLs, and state ground water standards. The chemicals which eceeded several of these ARARs and other guidance levels included benzene, chlorobenzene, 1,2-dichloroethane, trichloroethylene, chloroform, and many other VOCs, PCBs, total PAHs, phenol, arsenic, cadmium, chromium, lead, mercury and nickel.

TABLE E-2

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Soil	**Human Health Risks**	
	Excess lifetime cancer risks exceeded 1E-06 (one in one million) for trespassers who may contact on-site surface soil (average and reasonable maximum cases).	Aldrin, arsenic, carcinogenic PAHs, 1,1-dichloroethane, dieldrin, PCBs, tetrachloroethylene, trichloroethylene
	Under future site use conditions, excess lifetime cancer risks exceeded 1E-06 for possible future on-site workers who may contact surface soil (average and reasonable maximum cases).	Aldrin, arsenic, benzene, bis(2-ethylhexyl)phthalate, carcinogenic PAHs, 1,1-dichloroethane, 1,2-dichloroethane, dieldrin, PCBs, tetrachloroethylene, trichloroethylene
	Under future site use conditions, excess lifetime cancer risks exceeded 1E-06 for possible future on-site construction workers who may contact subsurface soil (reasonable maximum case).	Carcinogenic PAHs, PCBs
	Adverse noncarcinogenic effects may occur among trespassers who contact surface soil under current site use and among possible future on-site workers who contact surface soil (reasonable maximum cases only).	Aldrin, dieldrin, PCBs, tetrachloroethylene, trichloroethylene
	Ecological Risks	
	Not evaluated. The site is not currently a habitat for terrestrial wildlife or abundant plant life. The extent to which this is a result of site contamination could not be determined.	
	Migration Potential	
	Contaminants present in on-site soil have migrated downward to deeper soil depths, and into the water table and till aquifers. Further migration into the bedrock aquifer has also occurred. Chemicals may also be released from soil into air via volatilization and suspension or surface soil by wind or vehicles. Chemicals may migrate via surface soil runoff into Peach Island Creek.	VOCs, PAHs, PCBs
	Violations of ARARs/Other Guidance	
	NJDEP ECRA Objectives for soil	Total volatile organics (predominantly chlorobenzene, 1,1-dichloroethane, ethylbenzene, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene, xylenes)
		Total base neutrals/acids (predominantly PAHs, phenol, butyl benzyl phthalate, 1,2-dichlorobenzene)
		PCBs
		Inorganics (predominantly cadmium, copper, lead, mercury, zinc)
	TSCA PCB Spill Policy	PCBs

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

TABLE E-2 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Ground Water (Water Table and Till Aquifers)	**Human Health Risks**	
	Excess lifetime cancer risks for a possible future on-site worker, assumed to regularly consume on-site ground water, were greater than 1E-06 (one in one million) for both the water table and till aquifers for the average and reasonable maximum cases.	Arsenic, benzene, bis(2-ethylhexyl)phthalate, carcinogenic PAHs, chloroform, 1,1-dichloroethane, 1,2-dichloroethane, 1,1-dichloroethylene, isophorone, methylene chloride, PCBs, 1,1,2,2-tetrachloroethane, tetrachloroethylene, trichloroethylene, vinyl chloride
	Adverse noncarcinogenic effects may occur for a possible future on-site worker assumed to regularly consume on-site ground water for both the water table and the till aquifers (average and reasonable maximum cases).	Arsenic, chlorobenzene, chloroform, 1,2-dichloroethane, 1,2-trans-dichloroethylene, lead, methylene chloride, methyl ethyl ketone, nitrobenzene, PCBs, 1,1,2,2-tetrachloroethane, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene
	Ecological Risks	
	Not evaluated; no aquatic or terrestrial wildlife species are expected to come into contact with on-site ground water.	
	Migration Potential	
	Chemicals present in the water table and till aquifers may migrate into Peach Island Creek and to deeper soil depths. The water table aquifer flow is not well defined although it appears to flow radially across the site's boundaries and downward to the till aquifer. The till aquifer appears to flow towards the northwest. Further vertical migration into the bedrock aquifer has also occurred.	VOCs, PCBs
	Violations of ARARs/Other Guidance	
	Federal MCLs and MCLGs	Benzene, 1,2-dichloroethane, 1,1-dichloroethylene, ethylbenzene, 1,1,1-trichloroethane, chloroform, chlorobenzene, trichloroethylene, vinyl chloride, arsenic, cadmium, chromium, lead, mercury
	State MCLs	Benzene, chlorobenzene, chloroform, 1,1-dichloroethylene, 1,2-dichloroethane, 1,1,1-trichloroethane, methylene chloride, 1,2-trans-dichloroethylene, xylenes, tetrachloroethylene, trichloroethylene, vinyl chloride, arsenic, cadmium, chromium, lead, mercury, PCBs
	State Ground Water Quality Standards	Benzene, chlorobenzene, chloroform, 1,2-dicholroethane, 1,1-dichloroethylene, 1,2-trans-dichloroethylene, methylene chloride, tetrachloroethylene, 1,1,1-trichlroethane, trichloroethylene, vinyl chloride, xylenes, PCBs, phenol, arsenic, cadmium, chromium, copper, lead, mercury
	Federal Ambient Water Quality Criteria for Protection of Human Health (adjusted for drinking water only)	Benzene, 1,2-dichloroethane, 1,1-dichloroethylene, 1,1,2,2-tetrachloroethane, chlorobenzene, 1,1,1-trichloroethane, chloroform, ethylbenzene, tetrachloroethylene, trichloroethylene, toluene, vinyl chloride, PCBs, phenol, arsenic, beryllium, cadmium, chromium, copper, lead, nickel

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations

TABLE E-2 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Ground Water (Bedrock aquifer)	**Human Health Risks**	
	Excess lifetime cancer risks for a possible future on-site worker, assumed to regularly consume on-site bedrock ground water, were greater than 1E-06 (one in one million) for the average and reasonable maximum cases.	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, trichloroethylene, vinyl chloride
	Adverse noncarcinogenic effects may occur for a possible future on-site worker, assumed to regularly consume on-site groundwater from the bedrock aquifer (average and reasonable maximum cases).	Chloroform, trichloroethylene
	Ecological Risks	
	Not evaluated; no aquatic or terrestrial wildlife species are expected to come into contact with on-site ground water.	
	Migration Potential	
	Chemicals present in the bedrock aquifer may migrate to off-site locations from which water may be used as a public water supply.	VOCs
	Violations of ARARs/Other Guidance	
	Federal MCLs and MCLGs	Chloroform, 1,2-dichloroethane, trichloroethylene, vinyl chloride
	State MCLs	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, methylene chloride, tetrachloroethylene, trichloroethylene, vinyl chloride
	State Ground Water Quality Standards	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, methylene chloride, tetrachloroethylene, trichloroethylene, vinyl chloride
	Federal Ambient Water Quality Criteria for Protection of Human Health (adjusted for drinking water only).	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, tetrachloroethylene, trichloroethylene, vinyl chloride

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

TABLE E-2 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Surface Water	**Human Health Risks**	
	Not evaluated due to limited sampling data. Peach Island Creek near the site is, however, currently accessible to some extent. Since access could change in the future, this pathway should be evaluated if more data become available in the future.	
	Ecological Risks	
	Adverse effects to aquatic life may occur from short- and long-term exposure to concentrations of inorganic chemicals in Peach Island Creek.	Copper, mercury, nickel, zinc
	No adverse effects are expected to occur in mammalian wildlife (such as muskrats) through ingestion of surface water.	
	Migration Potential	
	Chemicals in Peach Island Creek may be transported both up and down stream although the magnitude of impact of the site is difficult to determine due to complex tidal nature of the creek and availability of only limited sampling results.	Ethylbenzene, xylenes, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene, PCBs, dieldrin, bis(2-ethylhexyl)phthalate, 1,2-dichlorobenzene
	Exceedances of ARARs/Other Guidance	
	State Surface Water Quality Standards	PCBs

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

TABLE E-2 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Sediment	**Human Health Risks**	
	Not evaluated.	
	Ecological Risks	
	Adverse effects to aquatic life may occur from short- and long-term exposure to inorganic and organic chemicals in sediments.	Dieldrin, PCBs, cadmium, copper, lead, mercury, zinc.
	Adverse effects may occur in water fowl (including endangered species) by ingesting contaminated invertibrates. There are considerable uncertainties (e.g., in calculated interstitial water concentrations and bioconcentration factors) in these estimates.	Dieldrin, cadmium, copper, lead, mercury, nickel, zinc.
	Migration Potential	
	Chemicals on-site have migrated into Peach Island Creek sediment, although the magnitude of impact is difficult to determine due to complex tidal nature of the creek and availability of only limited sampling results.	Ethylbenzene, xylenes, tetrachloroethylene, toluene, 1,1-trichloroethane, trichloroethylene, PCBs, dieldrin, bis(2-ethylhexyl)phthalate, 1,2-dichlorobenzene.
	Violations of ARARs/Other Guidance	
	Proposed NOAA sediment action level for protection of aquatic life.	PCBs

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

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TABLE E-2 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Air	**Human Health Risks** ------------------- Excess lifetime cancer risks for nearby workers who may inhale volatilized organics and suspended soil transported from the site exceeded 1E-06 (one in one million) (reasonable maximum case only). Adverse noncarcinogenic effects are not expected to occur.	Chromium, 1,1-dichloroethylene, vinyl chloride
	Excess lifetime cancer risk for possible future on-site workers who may inhale volatilized organics and suspended soil exceeded 1E-06 (one in one million) for both the average and reasonable maximum cases. Adverse noncarcinogenic effects are not expected to occur.	Chloroform, chromium, 1,1-dichloroethylene, methylene chloride, trichloroethylene, vinyl chloride
	Ecological Risks ---------------- Not evaluated; no terrestrial mammals observed on-site. Ambient air exposures are not likely to result in significant exposures.	
	Migration Potential ------------------- Chemicals released into air from the site (via volatilization and suspension of surface soil) may migrate off-site.	VOCs, PAHs, PCBs
	Violations of ARARs/Other Guidance ---------------------------------- Not available except for lead and its ARAR was not exceeded.	

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

For the quantitative assessment of risks, exposure estimates were combined with the health criteria for the selected chemicals of concern to estimate potential risks to human health. As for exposures, risks are estimated for an average and a RME case. The average case combines the average case exposure estimates with generally upper bound slope factors (for carcinogenic effects) and conservatively derived reference doses (for noncarcinogenic effects). The average case is intended to represent the exposure of a typical individual; however, use of conservative health criteria may result in an overestimation of risk even for the average case. The RME case combines the RME exposure estimates with generally upper bound slope factors and conservative reference doses. This scenario is intended to place a conservative upper bound on the potential risks.

The risks reported in this RA are estimates of current or potential risks to human health under the average or RME exposure pathways evaluated.

Table E-2 summarizes the risk estimates for the exposure pathways evaluated in this RA. In this table, the pathways for which the total potential upper bound lifetime excess cancer risks across all chemicals exceeded 1×10^{-6} (one in one million) are identified. An upper bound excess lifetime cancer risk of 1×10^{-6} means that an individual's incremental chance of developing cancer over a 70-year lifetime due to the specific exposure conditions evaluated is one in one million at most. The USEPA's target risk range for evaluating Superfund sites is from 1×10^{-6} (one in one million) to 1×10^{-4} (one in ten thousand). Also listed in this table are those individual chemicals for which the upper bound excess lifetime cancer risks exceeded 1×10^{-6}. The exposure pathways for

which total excess lifetime cancer risks across chemicals exceeded 1x10^{-6} were

as follows:

(1) Trespassers who may contact on-site surface soil (average and reasonable maximum cases). The risks were predominantly due to exposure to aldrin, arsenic, carcinogenic PAHs, 1,1-dichloroethane, dieldrin, PCBs, tetrachloroethylene, and trichloroethylene.

(2) Possible future on-site workers who may regularly contact surface soil (average and reasonable maximum cases). The risks were predominantly due to exposure to aldrin, arsenic, benzene, bis(2-ethylhexyl)phthalate, carcinogenic PAHs, tetrachloroethylene, 1,1-dichloroethane, 1,2-dichloroethane, dieldrin, PCBs, tetrachloroethylene, and trichloroethylene.

(3) Possible future on-site construction workers who may contact subsurface soil (reasonable maximum case only). The risks were predominantly due to exposure to carcinogenic PAHs and PCBs.

(4) Possible future on-site workers who may regularly consume ground water from the on-site water table, till, and bedrock aquifers. It should be noted that, for the water table and till aquifers, this pathway is unlikely to occur since these aquifers are not known to be used for water supply in the area. These aquifers were evaluated, however, because of the likelihood of migration from these aquifers to the bedrock aquifer which is used for drinking water in the area. The risks from use of the water table and till aquifer ground water for potable uses were associated with exposure to numerous volatile organic compounds, carcinogenic PAHs, and PCBs. The risks from use of the bedrock aquifer ground water were associated with exposure to volatile organic compounds.

(5) Workers currently employed near the site who may inhale volatilized organics and suspended soil released into the air (reasonable maximum case only). The risks were predominantly due to exposure to chromium, 1,1-dichloroethylene, and vinyl chloride.

(6) Possible future on-site workers who may inhale volatilized chemicals or suspended on-site soil. The risks were predominantly due to chloroform, chromium, 1,1-dichloroethylene, methylene chloride, trichloroethylene, and vinyl chloride.

Table E-2 also indicates which exposure pathways may result in adverse

noncarcinogenic effects. For the following pathways, there is a potential for

such adverse effects to occur:

(1) Trespassers who may contact on-site surface soil (reasonable maximum case only). These risks were predominantly due to aldrin, dieldrin, PCBs, tetrachloroethylene, and trichloroethylene.

(2) Possible future on-site workers who may regularly contact surface soil (reasonable maximum case only). These risks were predominantly due to exposure to aldrin, dieldrin, PCBs, tetrachloroethylene, and trichloroethylene.

(3) Possible future on-site workers who may regularly consume on-site ground water from the water table and till aquifers (although this pathway is unlikely to occur). These risks were predominantly due to numerous volatile organic compounds, arsenic, nitrobenzene, and PCBs. Adverse noncarcinogenic effects could also occur from regular ingestion of bedrock aquifer groundwater, primarily due to trichloroethylene and chloroform.

E.4 ECOLOGICAL RISK ASSESSMENT

Potential risks to environmental receptors were also evaluated for the SCP site using the results of site monitoring data, a review of the toxicity of the chemicals of concern, and estimates of exposure. Risks have been characterized by comparing chemical concentrations with federal criteria and by comparing estimated exposures with toxicity values provided in the scientific literature.

The potential risks from exposures of aquatic and terrestrial wildlife to chemicals in surface water and sediments of Peach Island Creek were assessed. The site is not currently a habitat for terrestrial wildlife or plants (with the exception of some sparse vegetation). Thus, since these ecological exposure pathways are not currently considered complete, they were not evaluated in this risk assessment. However, the absence of terrestrial wildlife and abundant plant life may be due, in part, to contamination at the site. The results of the environmental risk assessment are summarized below.

The potential risks to aquatic life were evaluated by comparing surface water
and sediment chemical concentrations with USEPA ambient water quality criteria
(AWQC), sediment quality criteria (SQC), and other toxicity values. It was
concluded that risks to aquatic life may occur from exposure to chemicals in
surface water since ambient water quality criteria were exceeded for copper,
mercury, nickel, and zinc. Risks to aquatic life from chemicals in sediments
are also likely to occur since: (1) dieldrin and PCB concentrations exceed
sediment quality criteria, (2) copper concentrations in sediments exceed
concentrations that were shown to be lethal in short-term laboratory tests
with invertebrates, and (3) concentrations of cadmium, lead, mercury and zinc
exceed levels that have produced biological effects in sediment toxicity tests
conducted in west coast estuaries. In addition, PCB concentrations in
sediments exceed the proposed 0.1 mg/kg preliminary action level for
protection of aquatic life by factors of 52 to 550. These comparisons imply
that populations of aquatic life that may inhabit Peach Island Creek may be at
risk. Thus it is likely that adverse effects are occurring from exposure of
aquatic life to contaminants in surface water and sediments.

Additionally, potential risks to the Pied-billed Grebe, which has an
endangered breeding population in the Meadowlands not far from Berry's Creek,
were evaluated. Estimated dietary concentrations (from consumption of
invertebrates in which sediment contaminants have bioaccumulated) of dieldrin,
cadmium, copper, lead, mercury, nickel, and zinc exceed toxicity values
derived from toxicological studies with birds. There are considerable
uncertainties associated with these estimated dietary concentrations since
they are based on: (1) limited sediment sampling data, (2) estimation of

sediment pore water concentrations by multiplying sediment concentrations by a
sediment:water partition coefficient, and (3) use of short-term invertebrate
bioconcentration factors. These data indicate, however, that adverse effects
to this endangered breeding population might occur should the population exist
near the site or feed extensively from Peach Island Creek. Birds that
frequent other, less contaminated areas for a portion of their diet, would be
at less risk.

It is unlikely that mammalian wildlife such as the muskrat will suffer adverse
effects from the ingestion of contaminants in surface water. Estimated doses
were at least one order of magnitude below toxicity values for all chemicals
of concern. Other possible routes of exposure for mammalian wildlife, such as
through the diet or contact with sediments, have not been quantified because
currently available data are insufficient to estimate doses or dietary levels.

E.5 CONCLUSIONS

Based on the results of this Baseline Risk Assessment it can be concluded that
the SCP site has caused severe degradation of the water table and till
aquifers at the site. The concentrations of numerous chemicals in the water
table and till aquifers exceed both federal and state drinking water standards
and guidelines. Contaminants at the site have also migrated into the bedrock
aquifer which is used as a public water supply source. Concentrations of
several site-related chemicals in the bedrock aquifer exceed both state and
federal drinking water standards and guidelines. In addition, contaminants
from the site are migrating into Peach Island Creek. The concentrations of
several chemicals in surface water and sediments of this creek exceed levels

associated with adverse effects in aquatic life, although it is not possible (at present) to definitively determine the extent of a site-related impact. Soil contact by site trespassers and future on-site workers could result in adverse human health impacts under the exposure scenarios evaluated. Furthermore, the concentrations of several chemicals in on-site soils exceed available state and federal guidelines.

1.0 INTRODUCTION

The Scientific Chemical Processing (SCP) site is currently the subject of a Comprehensive Environmental Response, Compensation and Liability Act (CERCLA, as amended), Remedial Investigation/Feasibility Study (RI/FS). The site is classified as an enforcement lead site for which potentially responsible parties (PRPs) are conducting the RI/FS. The PRPs for this site retained the engineering firm, Dames and Moore, to perform a detailed Remedial Investigation (RI).

This baseline risk assessment (BRA) was prepared at the request of Region II of the U.S. Environmental Protection Agency (USEPA) under the REM III Superfund contract. It has been conducted independently of the PRPs' efforts, but relies on the Dames and Moore (1990) RI report as the primary source of information concerning conditions at the site.

A BRA estimates the potential magnitude and probability of actual or potential harm to public health and the environment caused by threatened or actual releases of hazardous substances into the environment. This BRA addresses the potential human health and environmental impacts associated with the SCP site under the no-action alternative, that is, in the absence of remedial (corrective) action as required under Section 300.68(f)(v) of the National Contingency Plan.

The procedures used in this BRA follow USEPA guidance for risk assessments in general (USEPA 1986b,c,d), and for Superfund sites in particular (USEPA 1989a). Although this latter guidance, the Interim Final Risk Assessment Guidance for Superfund: Human Health Evaluation Manual, was released during the course of this project, it has been followed, at the request of USEPA Region II, to the extent possible given the time-frame of this project (e.g., see Section 3). In this guidance, USEPA states that the baseline risk assessment should evaluate reasonable maximum exposures (RME) (USEPA 1988) expected to occur under both current and future land-use conditions. USEPA (1989a) notes, "the intent of the RME is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures."

The majority of the sampling data used in this BRA were collected and analyzed as part of the Dames and Moore (1990) RI. In addition, two samples collected from the bedrock aquifer in 1989 and analyzed by USEPA during continuation of the Dames and Moore RI work were used. The Dames and Moore (1990) RI data were obtained from the raw laboratory data reports (ETC December 1987) provided to USEPA by Dames and Moore. These data were also summarized (ETC February 1988) in the Dames and Moore (1990) RI in a series of appendices. For the purposes of this assessment, and at the request of Region II USEPA, the December 1987 raw laboratory reports only (laboratory validated) have been used.

This BRA is organized as follows: In Section 2, a summary of the site history is presented to provide a framework for this assessment. Chemicals of concern considered most likely to pose risks to human health are then identified for each environmental medium sampled. In Section 3, Exposure Assessment, the migration potential of the selected chemicals of concern is evaluated through examination of site environmental factors, waste characteristics and physical and chemical properties of the selected chemicals. Then potential exposure pathways under current and hypothetical future site use conditions are identified and concentrations of the chemicals of concern at potential exposure points are estimated. In Section 4, the hazard assessment, the health criteria (i.e., dose-response) values used in the quantitative estimation of potential health risks are identified. In addition, the range of potential health effects for each of the chemicals of concern is briefly reviewed. In Section 5, the estimated exposure point concentrations given in Section 3 are compared to applicable or relevant and appropriate requirements (ARARs) where they exist for the chemicals of concern. Then, for each identified exposure pathway which will be quantitatively evaluated, potential chemical intakes are estimated and combined with the health criteria values to predict potential human risks. In Section 6, an ecological risk assessment is presented for the site. Section 7 presents a discussion of uncertainties in the BRA. Section 8 presents the summary and conclusions of this assessment.

2.0 SITE CHARACTERIZATION

This section reviews the history of the SCP site and briefly describes the current site condition including the results of the RI performed by Dames and Moore (1990). Key site features are discussed to provide a framework for the EA, and then chemicals of concern for detailed evaluation in this assessment are identified.

2.1 SITE HISTORY

The SCP site is located in Carlstadt Township, Bergen County, New Jersey (see Figure 1). The SCP site was reportedly used for solvent refining and solvent recovery. Aerial photographs (dates unreported) indicated the presence of drums at the site. The property is owned by Inmar Associates which leased the site to Scientific Chemical Processing, Inc. (SCP) in October 1970. SCP used the site for recycling industrial wastes such as liquid hydrocarbons and liquid byproduct streams from chemical and other industrial manufacturing firms. The materials were processed to marketable products such as methanol, or were blended and sold as boiler fuel (Dames and Moore 1990).

Dames and Moore (1990) states that the site also received other materials such as paint sludges and acids, until at least 1980. Over 300,000 gallons of material stored on the site, primarily in liquid form, were removed from the site during the early 1980s. These wastes included fuel, fuel residue and water mixtures, methanol/phosphoric acid solutions, etching solutions,

LOCATION MAP
SCP SITE
CARLSTADT, NEW JERSEY

FIGURE 1

```
0          2000          4000
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SCALE IN FEET

2-2

solvents, and thinners. In 1986, four (4) tanks containing PCB-contaminated materials were removed from the site by Inmar Associates, pursuant to an Administrative Order issued by USEPA.

During SCP's operations, equipment and material at the site included a tank farm, a still and boiler house, and a staging platform and thin-film evaporator. Figure 2, reproduced from the Dames and Moore (1990) RI, indicates that an area on the eastern side of the site was a former sludge pit. Unlined drum storage areas without spill containment provisions were located on the southeastern portion of the site (Dames and Moore 1990).

The tank farm area was not paved and presently contains a tank containing PCBs and other hazardous substances. Its structural integrity is suspect because streaks of discoloration appear on the sides of the tank and patches are visible (Dames and Moore 1990). Tanks and tank trailers were also reported to have been removed from the section of the site that contained the still and boiler house. Discoloration of the soil was reported (Dames and Moore 1990) in these areas. The site contained a staging platform that was used for transferring and storing wastes and also included a sludge disposal area.
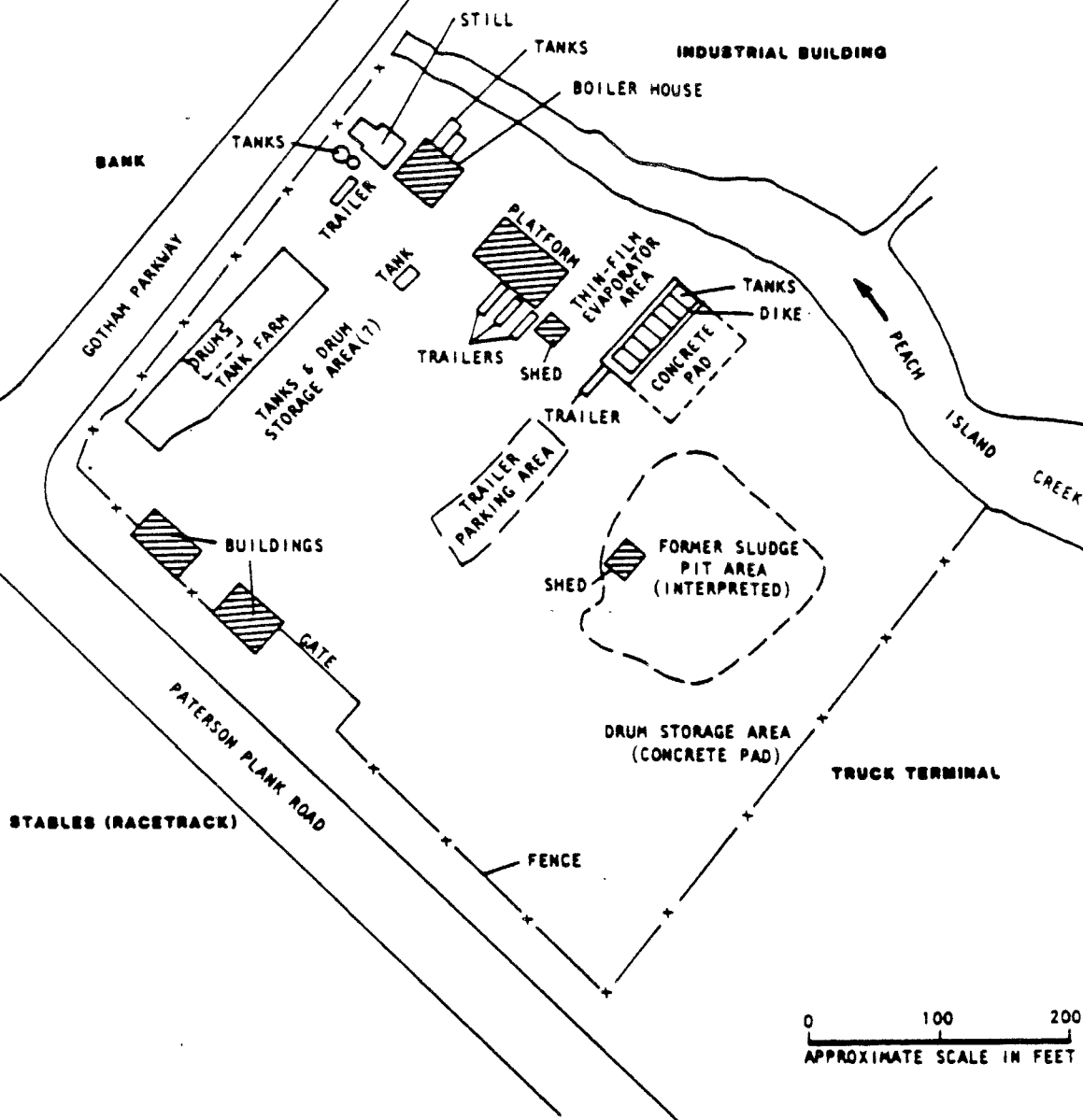
SITE LAYOUT
SCP SITE
CARLSTADT, NEW JERSEY

NOTES:
1. ALL DRUMS, MOST TANKS AND TANK TRAILERS HAVE BEEN REMOVED AND SOME FACILITIES HAVE BEEN DISMANTLED SINCE OPERATIONS CEASED IN 1979.
2. BASE MAP REFERENCE: AERIAL PHOTOGRAPH NO. 3818-6-35, MARCH 27, 1984.

FIGURE 2

2.2 ENVIRONMENTAL CHARACTERIZATION

2.2.1 Setting and Land Use

The SCP site is located on 5.9 acres of flat, sparsely-vegetated land, with vegetation appearing more commonly along the periphery of the site. The site is located at the corner of Paterson Plank Road and Gotham Parkway, directly across from the Meadowlands Racetrack and Stables. The site is bounded by Peach Island Creek on the northeast and enclosed by a locked chain link fence on the remaining three sides (see Figure 2). The surface of unvegetated areas has the appearance of gravel, and a few rusted drums and one tank are visible on site.

Businesses and commercial facilities are located in the immediate vicinity of the site, including a bank, a truck terminal, industrial facilities, a new office building that is at least partially occupied, and an indoor racket club. Nearby industries include a leather company and a metal casting corporation.

Current zoning by New Jersey's Hackensack Meadowlands Development Commission (HMDC) is classified as B; light industry and distribution. In addition to light industry, hotels and restaurants are permitted as "special exceptions". The HMDC is re-examining the land use master plan in a two-year project expected to be completed in 1990.[1] Based on the Dames and Moore (1990)

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[1] W. Nierstedt, Engineer. HMDC. Personal Communication. June 29, 1988.

report, the nearest residence is located roughly 5,000 feet from the site along Washington Avenue to the northeast of the site.

The SCP site is located in a wetland area. No standing surface water was observed at the site; the bare ground cover appears to be gravel. The salt water marshes and swamps in the site vicinity are drained by the Hackensack River and its tributaries one of which is Berry's Creek. One of the tributaries of Berry's Creek is Peach Island Creek, which flows along the north boundary of the site. Peach Island Creek is tidal with surface water levels fluctuating by up to roughly one foot as a result. The land lying along these creeks has been classified as a waterfront recreation zone (Dames and Moore 1990). The far bank of Peach Island Creek adjacent to the site is currently bounded by a parking lot for an industrial facility.

2.2.2 Geology/Hydrogeology

A detailed description of the site geology including information on stratigraphy was reported in the Dames and Moore (1990) RI (Section 4.1). The following information is briefly summarized from that report.

The site is located within the Piedmont Geologic Province of New Jersey underlain by shales and sandstones. The site contains heterogeneous man-made fill mixed with soil ranging from 3 to 11 feet below the surface. This area is underlain, in descending order, by peat (0-7 feet thick), gray silt (roughly 2 feet thick), varved clay (0-18 feet thick, beginning on average

approximately 8-10 feet below the site's surface), till (approximately 20 feet thick) and bedrock (Brunswick shale). An example of the site's stratigraphy is shown in a composite cross-section in Figure 3, reproduced from Dames and Moore (1990). This cross-section is from location A to A' shown on Figure 4 (see Section 2.3). The bedrock was not studied during the Dames and Moore (1990) RI; soil sampling only extended to the bottom of the clay layer. Subsequently, as part of Dames and Moore's ongoing RI work, additional ground water data were collected and analyzed by USEPA, including two samples from the bedrock aquifer collected in February and April 1989. The resulting data are used in this EA.

The three aquifer systems at the site are the shallow water table aquifer (starting at 1-2 feet below the surface and extending down to the clay layer), the glacial till aquifer (approximately 25-50 feet deep, located between the clay and the underlying aquifer), and the deep bedrock aquifer. The water table and till aquifers are separated by a layer of clay and silt. Ground water flow patterns of both the water table and till aquifers are discussed in the Dames and Moore (1990) RI (Sections 4.3.2 and 4.3.3). The ground water flow pattern in the water table aquifer was poorly defined in the RI, although the report indicated that ground water in this aquifer was migrating away from the site in a horizontal direction across all borders of the site. The flow velocities and flow rates associated with ground water movement in the shallow water table aquifer were not provided to USEPA in the Dames and Moore (1990) RI. Dames and Moore has recently indicated that the dominant flow in the

FIGURE 3

CROSS-SECTION A-A'

SCP SITE
CARLSTADT, NEW JERSEY
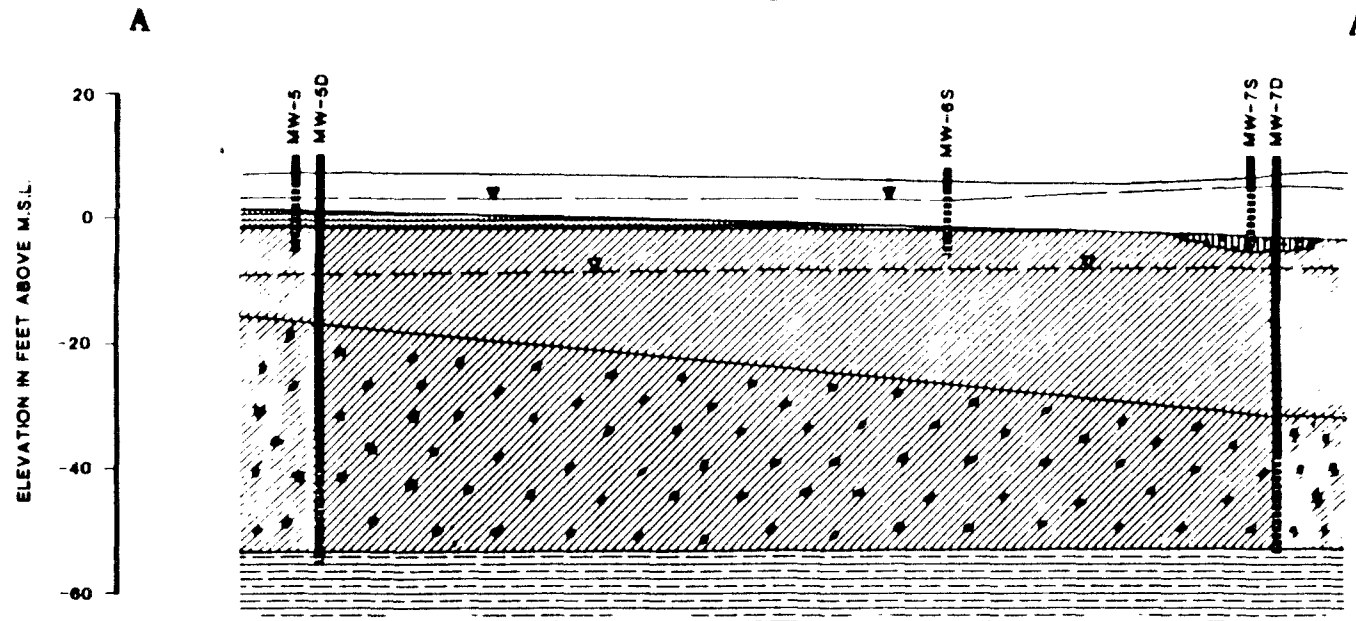
KEY:

MISCELLANEOUS FILL

PEAT

GRAY SILTY FINE SAND

GRAY & BROWN CLAYEY SILT
& CLAY (LACUSTRINE)

REDDISH BROWN MIXTURE OF
CLAY, SILT, SAND & GRAVEL
(GLACIAL TILL)

REDDISH BROWN BRUNSWICK SHALE

SCREENED INTERVAL

WATER TABLE SURFACE

POTENTIOMETRIC SURFACE

HORIZONTAL SCALE IN FEET
2.5 x VERTICAL EXAGGERATION

NOTE:
THE SUBSURFACE SECTION SHOWN REPRESENTS OUR
EVALUATION OF THE MOST PROBABLE CONDITIONS
BASED UPON INTERPRETATION OF PRESENTLY AVAIL-
ABLE DATA.

shallow aquifer may be downward, across the clay-silt layer, into the till aquifer.

The water table aquifer does not appear to be influenced by the tides experienced by Peach Island Creek, possibly due to the fact that the water table under the site is approximately five feet higher than the water surface elevation in the creek. Given this five foot head differential, it is highly probable that the ground water in the water table aquifer is discharging directly into Peach Island Creek. The water table aquifer does respond rapidly to precipitation events, which is not suprising given the shallowness of this aquifer (approximately two feet below grade) and the high permeability of the soil-fill mixture located above the clay layer at the site.

The till aquifer is overlain by a clay/silt unit above (with weathered bedrock below). Ground water in the till aquifer flows in a northwesterly direction and is also subject to tidal influences (dissimilar to those observed in Peach Island Creek). Based on measurements taken by Dames and Moore (1990), a downward hydraulic gradient exists between the water table and till aquifers. The site sampling data strongly suggest that hazardous substances have migrated from the shallow aquifer into the till aquifer, and from the soil-fill mixture downward into the clay layer [e.g., see Figure 24 of the Dames and Moore (1990) report].

2.2.3 Climatology

The climate and meteorological conditions at the site have been characterized using information from Newark International Airport, which is approximately nine miles southeast of the site. The airport is in a similar setting as the site and therefore is considered to be representative of the site.

Climate in the site area includes moist, warm summers and moderately cold winters. Wind rose diagrams were included in the RI (Dames and Moore 1990) and indicate that winds in the area blow predominantly from the southwest with small seasonal variations in direction. Precipitation falls fairly uniformly throughout the year, and annual average precipitation is approximately 42 inches; seasonal tropical storms and hurricanes do occur. Dames and Moore (1990) state that the average annual precipitation of 42 inches minus the average annual potential evaporation of 35 inches results in a net annual precipitation of approximately 7 inches which, in theory, is the net amount of water available for ground water recharge and surface runoff. In any case, the site receives an influent average annual precipitation of approximately seven million gallons, which is subsequently released from the site in ground water flow and surface runoff from the site, and in the form of evapotranspiration into the atmosphere.

2.3 IDENTIFICATION OF CHEMICALS OF CONCERN

The Dames and Moore (1990) RI report discusses and contains the detailed

results of the site investigation. The results of sampling performed during

the RI are summarized in this section to identify chemicals to be evaluated in

detail in this risk assessment (chemicals of concern).

Data are summarized for each environmental medium by presenting frequencies of

detection, concentration ranges identifying the maximum detected values, and

geometric means[2] of each chemical. The following guidelines were used in

summarizing the data:

o   The data used to evaluate the SCP site were provided by Dames and
    Moore (1990) as part of a Remedial Investigation (RI) under contract
    to the potentially responsible parties for the site. Two sampling
    rounds were conducted by Dames and Moore (1990) in July and December
    1987. With the exception of the ground water data, the July 1987
    sample results were determined to be invalid (Dames and Moore 1990)
    and were therefore not used in this analysis. In addition, samples
    collected in February and April 1989 from the bedrock aquifer and
    analyzed by USEPA were also used (no bedrock data were available from
    the Dames and Moore 1987 sampling rounds).

o   To calculate the geometric mean for a medium in which a chemical was
    positively detected in more than one sample, non-detects were
    included in the mean by using one-half of each sample-specific
    detection limit. Where sample-specific detection limits were
    unavailable, one-half of USEPA's Contract-Required Quantitation Limit
    (CRQL) was used. This arbitrarily selected value (one-half) is
    commonly assigned to non-detects when averaging data for risk
    assessment purposes, since the actual value can be between zero and a
    value just below the detection limit (Vollmerhausen and Turnham

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[2]Geometric means rather than arithmetic means were calculated because
collections of environmental concentrations tend to be log-normally
distributed (Dean 1981, Esmen and Hammad 1977, Ott 1988).

1988). A geometric mean concentration was not calculated for those chemicals detected in only one sample because the mean would not be considered representative of the average chemical concentration.

o   Concentrations reported for duplicate samples at a given sampling point were averaged by calculating a geometric mean of the sample and its associated duplicate(s). This value was used in the analysis unless it was below the sample detection limit in which case the sample was treated as a non-detect.

The extent of contamination in the areas sampled and the identification of site-related chemicals by medium sampled are presented in the following sections. Based on the summarized data, chemicals are selected for further evaluation in the risk assessment. The criteria for selection included frequency of detection, detection in more than one environmental medium, concentrations detected, toxicity of the chemicals, and presence in environmental media at concentrations above background concentrations (for inorganics in soil). Sample concentrations of inorganic chemicals were compared with those levels considered to be naturally occurring in soil in order to determine if the detected levels were elevated above background. In comparing inorganic results with naturally occurring levels, statistical evaluation was not possible since sufficient numbers (three or more) of background samples were not available to calculate the standard deviation needed for an appropriate statistical test. No background samples were taken during the RI. However, a comparison of SCP site soil results with available regional soil background values was made. Sample levels which were within these background ranges were considered to be present at naturally occurring levels and were not further evaluated. Available regional background data of inorganic chemicals in soil are presented in Table 2-1.

TABLE 2-1

BACKGROUND SOIL CONCENTRATIONS FOR INORGANIC CHEMICALS
IN SCP SITE REGION

Chemical	Range of Concentrations (mg/kg)[a]		
Antimony	1.3	-	10
Arsenic	8	-	13
Beryllium	2	-	15
Cadmium[b]	<1	-	1
Chromium	100	-	1,000
Copper	50	-	700
Lead	50	-	700
Mercury	0.042	-	0.066
Nickel	30	-	700
Selenium	<0.1	-	0.1
Silver	NA		
Zinc	120	-	3,500

[a]Source: Based on surface soil data collected in Sussex
and Somerset Counties, New Jersey (Shacklette
and Boerngen 1984).

[b]Based on B Horizon Eastern U.S. Soils (Connor and
Shacklette 1975).

NA = Not available.

Chemicals which were detected infrequently in the samples collected from a specific medium (e.g., roughly 5%) and/or were detected at low concentrations [near to or below USEPA's Contract-Required Quantitation Limits (CRQLs)] in a sampled environmental medium, and are not known to be associated with past disposal practices are not considered to be site-related chemicals of concern. In addition, chemicals which are essential human nutrients (e.g., copper) and are unlikely to pose risks to human health at the levels observed on site were also not selected for detailed analysis.

Figure 4 illustrates the SCP site locations sampled by Dames and Moore (1990) for soil and ground water.

2.3.1 <u>Soil</u>

Soil samples were collected and analyzed at 17 locations at the SCP site. The samples were collected from four different depth ranges from the surface down to the clay layer. Shallow soil samples were collected in the unsaturated fill from 0-2 feet in depth, medium depth soil samples were collected in the saturated fill from 5-6 feet, deep soil samples were collected at the top of the clay layer, and very deep samples (collected only at 3 of the 17 locations) were collected at various depths within the clay layer below the site. All soil samples were analyzed for priority pollutants and some additional compounds. Analyses showed the presence of a wide variety of organic and inorganic chemical contaminants to be present in the site soil. The results are discussed below by depth range.

FIGURE 4

LOCATION PLAN

BORINGS, PIEZOMETERS AND WELLS

SCP SITE, CARLSTADT, N.J.
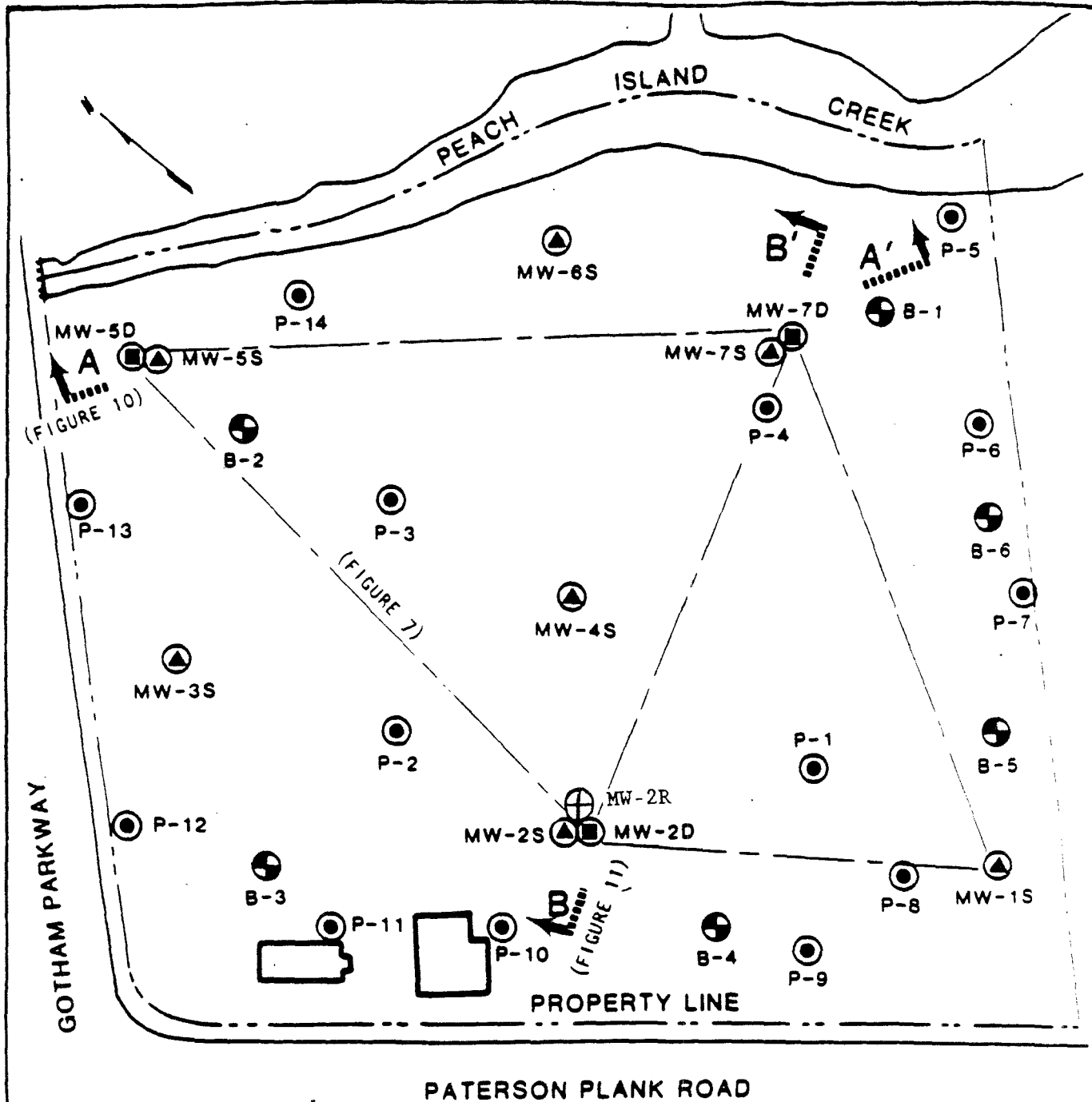
NOTE: REPLACEMENT BORINGS WITH PREFIX "R" WERE DRILLED ADJACENT TO ORIGINAL BORING LOCATIONS. FOR CLARITY THE "R" BORINGS ARE NOT SHOWN ON THIS FIGURE. SEE SECTION 4.2.1 FOR DISCUSSION.

KEY
- SOIL BORING
- SHALLOW PIEZOMETER
- SHALLOW MONITORING WELL
- DEEP MONITORING WELL
- BEDROCK MONITORING WELL

0   100   200 FEET
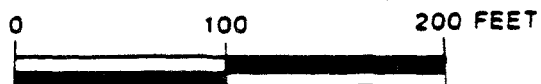
DAMES & MOORE

2.3.1.1 Shallow Soil (0-2') Samples

Table 2-2 summarizes the results of the sampling in shallow soil (0-2') at the
site. Eighteen volatile organic chemicals (VOCs) were detected in the surface
soil samples. Three of these (1,1,2,2-tetrachloroethane, 1,1,2-trichloro-
ethane and 1,1-dichloroethylene) were detected infrequently (i.e., in only one
or two of the total 17 samples analyzed) and were also detected infrequently
or were not detected at other depths (see the following sections). 1,1,2,2-
Tetrachloroethane and 1,1-dichloroethylene were, however, detected in ground
water (see Section 2.3.2) and thus were retained as chemicals of concern.
1,1,2-Trichloroethane was not detected in other environmental media and thus
will not be considered further. Although methyl ethyl ketone, 1,1-
dichloroethane, and 1,1,1-trichloroethane were also detected infrequently at
this depth, they were detected more frequently at other depths and were
detected in ground water; thus they were not removed from evaluation.

The VOCs remaining as chemicals of concern in soils are:

1,1,1-trichloroethane	ethylbenzene
1,1-dichloroethane	methyl ethyl ketone
1,2-dichloroethane	methylene chloride
1,2-trans-dichloroethylene	xylenes
benzene	tetrachloroethylene
chlorobenzene	toluene
chloroform	trichloroethylene
1,1-dichloroethylene	1,1,2,2-tetrachloroethane

Two chlorinated pesticides (aldrin and dieldrin) were detected in three and
five out of the 17 shallow soil samples, respectively. PCBs were detected in

2-16

TABLE 2-2

SUMMARY OF CHEMICAL CONCENTRATIONS
IN SHALLOW SOIL (0-2') SAMPLES

CHEMICAL (Concentration Units)	FREQUENCY OF DETECTION	MAXIMUM DETECTED CONCENTRATION	GEOMETRIC MEAN CONCENTRATION
Volatile Organic Compounds (ug/kg)			
Benzene	4/17	53,900	90
Chlorobenzene	4/17	336,000	128
Chloroform	4/17	17,800	44
1,1-Dichloroethane	2/17	64,700	72
1,2-Dichloroethane	4/17	10,200	60
1,1-Dichloroethylene	2/17	182	10
1,2-trans-Dichloroethylene	5/17	241	9
Ethylbenzene	7/17	652,000	384
Methyl ethyl ketone	2/17	8,560	104
Methylene chloride	11/17	2,390	143
1,1,2,2-Tetrachloroethane	1/17	476	NC
Tetrachloroethylene	12/17	4,290,000	934
Toluene	8/17	3,380,000	739
1,1,1-Trichloroethane	1/17	1,228	NC
1,1,2-Trichloroethane	2/17	1,810	31
Trichlorethylene	12/17	2,060,000	270
m-Xylene	7/17	2,000,000	734
o+p-Xylenes	9/17	1,450,000	825
Semi-Volatile Compounds (ug/kg)			
Acenaphthene (NC)	9/17	2,700	359
Anthracene (NC)	9/17	3,910	392
Benzo(a)anthracene (C)	5/17	4,540	1,040
Benzo(a)pyrene (C)	9/17	9,390	836
Benzo(b)fluoranthene (C)	6/17	17,700	1,990
Benzo(g,h,i)perylene (NC)	6/17	6,950	851
Benzo(k)fluoranthene (C)	1/17	3,790	NC
Bis-(2-ethylhexyl)phthalate	17/17	281,000	33,600
Butyl benzyl phthalate	8/17	48,304	1,540
2-Chloronaphthalene	2/17	122,000	174
Chrysene (C)	11/17	5,500	753
Dibenzo(a,h)anthracene (C)	3/17	2,400	693
1,2-Dichlorobenzene	8/17	47,300	543
2,4-Dichlorophenol	1/17	1,102	NC
2,4-Dimethylphenol	2/17	1,120	188
Diethylphthalate	1/17	4,994	NC
Di-n-butyl phthalate	13/17	71,000	3,080
Di-n-octyl phthalate	6/17	9,050	1,570
Fluoranthene (NC)	16/17	15,300	1,850
Fluorene (NC)	8/17	6,909	428
Indeno-(1,2,3-c,d)pyrene (C)	6/17	12,100	1,010
Naphthalene (NC)	16/17	102,000	2,020
Nitrobenzene	1/17	78,299	NC
N-Nitrosodiphenylamine	3/17	2,980	245
Phenanthrene (NC)	13/17	15,300	2,120
Phenol	4/17	58,200	145
Pyrene (NC)	15/17	12,700	1,800
1,2,4-Trichlorobenzene	2/17	1,228	152

TABLE 2-2 (Continued)

SUMMARY OF CHEMICAL CONCENTRATIONS
IN SHALLOW SOIL (0-2') SAMPLES

CHEMICAL (Concentration Units)	FREQUENCY OF DETECTION	MAXIMUM DETECTED CONCENTRATION	GEOMETRIC MEAN CONCENTRATION
Pesticides/PCBs (ug/kg)			
Aldrin	3/17	57,000	44
Dieldrin	5/17	57,000	170
PCBs:			
Aroclor 1242	11/17	15,000,000	2,680
Aroclor 1248	4/17	23,000	345
Aroclor 1260	2/17	48,000	351
Aroclor 1254	3/17	12,000	579
Inorganic Chemicals (mg/kg)			
Antimony	3/17	16	3.8
Arsenic	14/17	60	8.1
Beryllium	17/17	26	0.56
Cadmium	17/17	95.1	6.1
Chromium	17/17	721	78.5
Copper	17/17	71,600	2,320
Cyanide	16/17	5.02	1.85
Lead	17/17	2,750	490
Mercury	17/17	21.3	1.4
Nickel	15/17	39	12.2
Selenium	5/17	4.9	0.49
Silver	7/17	19	1.1
Zinc	17/17	4,170	398

ND = Not detected.
NC = Not calculated since chemical was detected in only one sample.

(C) = Carcinogenic PAH.
(NC) = Noncarcinogenic PAHs.

most of the shallow soil samples; for example Aroclor 1242 was detected in 11 of the 17 shallow soil samples analyzed. These three chemicals (aldrin, dieldrin, and PCBs) were retained for detailed analysis in this assessment.

In the semi-volatile chemical class, 28 chemicals were detected. As with a few of the VOCs, four semi-volatiles (1,2,4-trichlorobenzene, 2,4-dichloro-phenol, nitrobenzene, and diethylphthalate) were detected infrequently in shallow soil (i.e., in only one or two of the samples) and were also detected infrequently and/or were not detected at all the other soil depth ranges. Nitrobenzene was, however, selected as a chemical of concern in groundwater (see Section 2.3.2) and thus was also selected for soil. The other three organics were, however, removed from further evaluation.

N-nitrosodiphenylamine was detected in three of the 17 shallow soil samples, but was detected in only one of 17 medium depth soil samples (see later section), and not detected at all in the deep or very deep soil samples or other sampled media. Based on its infrequent detections, it was also removed from further analysis. 2,4-Dimethylphenol was detected in two of the shallow soil samples, three of the medium depth soil samples, one shallow sediment sample (upstream only), and in the water table aquifer. It was thus retained for further analysis.

Many of the detected semi-volatiles are members of a class of compounds collectively referred to as polycyclic aromatic hydrocarbons (PAHs). For the purposes of risk assessment, USEPA has recommended that PAHs be divided into

two general classes, carcinogenic PAHs and noncarcinogenic PAHs. Based on the International Agency for Research on Cancer (IARC) classification of PAHs, carcinogenic PAHs are those with sufficient or limited evidence of carcinogenicity in animals while noncarcinogenic PAHs are those for which evidence of carcinogenicity in animals is inadequate or negative (see Table 2-3). In Table 2-2 (as well as the subsequent soil tables in this section), the classification of each detected PAH (carcinogenic or noncarcinogenic) has been noted. Since PAHs of both classifications were detected, both carcinogenic and noncarcinogenic PAHs (CPAHs and NCPAHs, respectively) will be retained for further analysis in this assessment.

In addition to 2,4-dimethylphenol, CPAHs, and NCPAHs, seven other semi-volatiles were retained as chemicals of concern: 1,2-dichlorobenzene, 2-chloronaphthalene, bis(2-ethylhexyl)phthalate, butyl benzyl phthalate, di-n-butyl phthalate, di-n-octyl phthalate, and phenol. In summary, the semi-volatiles retained as chemicals of concern in soil are as follows:

bis(2-ethylhexyl)phthalate	1,2-dichlorobenzene
butylbenzyl phthalate	2,4-dimethylphenol
carcinogenic PAHs (7 CPAHs)	noncarcinogenic PAHs (8 NCPAHs)
2-chloronaphthalene	di-n-octyl phthalate
di-n-butyl phthalate	phenol

Thirteen inorganic chemicals were detected in the shallow soil samples. Among these, two (chromium and nickel) were detected in shallow soil at levels within the regional background concentration range and within the background range at the other soil depths as well. These chemicals are, however,

TABLE 2-3

IARC CLASSIFICATION OF PAHs ACCORDING TO
EVIDENCE FOR CARCINOGENICITY

Chemicals for which there is sufficient evidence that they are carcinogenic in animals:

Benzo(a)anthracene	7H-Dibenzo(c,g)carbazole
Benzo(b)fluoranthene	Dibenzo(a,e)pyrene
Benzo(j)fluoranthene	Dibenzo(a,h)pyrene
Benzo(k)fluoranthene	Dibenzo(a,i)pyrene
Benzo(a)pyrene	Dibenzo(a,l)pyrene
Dibenzo(a,h)acridine	Indeno(1,2,3-cd)pyrene
Dibenzo(a,h)anthracene	

Chemicals for which there is limited evidence that they are carcinogenic in animals:

Anthranthrene	Dibenzo(a,c)anthracene
Benzo(c)acridine	Dibenzo(a,j)anthracene
Carbazole	Dibenzo(a,e)fluoranthene
Chrysene	2-, 3-, 4-, and 6-Methylchrysene
Cyclopenta(c,d)pyrene	2- and 3-Methylfluoranthene

Chemicals for which the evidence is inadequate to assess their carcinogenicity:

Benzo(a)acridine	Coronene
Benzo(g,h,i)fluoranthene	1,2-Dimethylphenanthrene
Benzo(a)fluorene	Fluorene
Benzo(b)fluorene	1-Methylchrysene
Benzo(c)fluorene	1-Methylphenanthrene
Benzo(g,h,i)perylene	Perylene
Benzo(c)phenanthrene	Phenanthrene
Benzo(e)pyrene	Triphenylene

Chemicals for which the available data provide no evidence that they are carcinogenic:

Anthracene	Pyrene
Fluoranthene	Naphthalene[a]

Source: IARC (1983).

[a] Considered to be noncarcinogenic by USEPA.

retained as chemicals of concern because of their presence in other media
(surface water, sediments and ground water) for which they will be selected as
chemicals of concern (see later sections). Because soil background data were
unavailable for silver, it was retained as a chemical of concern.

Beryllium was detected in one shallow soil sample exceeding the background
range but at levels within the background range at other soil depths (see
later sections) and thus was not retained as a chemical of concern. Copper
was removed from further analysis because it is an essential human nutrient
and considered unlikely to be of concern to human health at the levels
observed on site. The 11 inorganic compounds remaining as chemicals of
concern in soil are thus:

antimony	lead
arsenic	mercury
cadmium	nickel
chromium	selenium
cyanide	silver
	zinc

2.3.1.2 Medium Depth (5-6') Soil Samples

Table 2-4 summarizes the results of the soil sampling in medium depths (5-6')
at the site. Eighteen VOCs were detected, and all but vinyl chloride were
also detected in the shallow soil samples. Although vinyl chloride was
detected infrequently (in only one of the 17 medium depth soil samples, not at
all in the shallow or very deep samples, and in one of the 17 deep samples),
it was still retained for evaluation in this analysis. This is because vinyl

TABLE 2-4

SUMMARY OF CHEMICAL CONCENTRATIONS
IN MEDIUM DEPTH (5-6') SOIL

Chemical (Concentration Units)	Frequency of Detection	Maximum Detected Concentration	Geometric Mean Concentration
Volatile Organic Compounds (ug/kg)			
benzene	8/17	52,300	621
chlorobenzene	7/17	258,000	887
chloroform	2/17	379,000	257
1,1 - dichloroethane	3/17	179,000	461
1,2 - dichloroethane	4/17	290,000	413
1,2 - trans-dichloroethylene	5/17	512,000	288
ethylbenzene	15/17	529,000	4,330
methyl ethyl ketone	5/17	795,000	1,300
methlyene chloride	8/17	14,900	565
1,1,2,2 - tetrachloroethane	1/17	703	NC
tetrachloroethylene	12/17	1,690,000	2,760
toluene	16/17	2,270,000	15,700
1,1,1 - trichloroethane	3/17	1,770,000	473
1,1,2, - trichloroethane	1/17	15,700	NC
trichlorethylene	8/17	1,670,000	856
vinyl chloride	1/17	28.9	NC
m-xylene	16/17	1,580,000	12,200
o+p - xylenes	16/17	710,000	10,500
Semi-Volatile Compounds (ug/kg)			
acenaphthene (NC)	8/17	21,200	443
acenaphthylene (NC)	1/17	21,000	NC
anthracene (NC)	7/17	2,950	474
benzidine	1/17	244,000	NC
benzo(a)anthracene (C)	5/17	84,200	1,200
benzo(a)pyrene (C)	7/17	108,000	649
benzo(b)fluoroanthene (C)	6/17	164,000	1,730
benzo(g,h,i)perylene (NC)	5/17	73,300	671
bis (2-ethylhexyl)phthlate	14/17	381,000	14,400
butylbenzylphthalate	6/17	73,600	1,990
2 - chloronaphthalene	4/17	18,200	282
chrysene (C)	7/17	106,000	633
1,2 - dichlorobenzene	6/17	385,000	499
diethyl phthalate	1/17	28,500	NC
2,4 - dimethylphenol	3/17	10,800	382
di-n-butyl phthalate	6/17	98,200	1,750
di-n-octyl phthalate	5/17	19,500	1,190
fluoranthene (NC)	13/17	176,000	1,460
fluorene (NC)	9/17	94,100	549

TABLE 2-4 (continued)

SUMMARY OF CHEMICAL CONCENTRATIONS
IN MEDIUM DEPTH (5-6') SOIL

Chemical (Concentration Units)	Frequency of Detection	Maximum Detected Concentration	Geometric Mean Concentration
Semi-Volatile Compounds (ug/kg) (continued)			
indeno(1,2,3-c,d)pyrene (C)	4/16	86,900	697
naphthalene (NC)	14/17	480,000	1,690
nitrobenzene	1/17	1,350,000	NC
N-nitrosodiphenylamine	1/17	157	NC
phenanthrene (NC)	9/17	268,000	1,960
phenol	4/17	790,000	405
pyrene (NC)	12/17	118,000	1,130
1,2,4 - trichlorobenzene	2/17	4,930	222
Pesticides/PCBs (ug/kg)			
aldrin	1/14	1,200	NC
dieldrin	3/13	940	23
methoxychlor	1/17	150,000	NC
PCBs:			
Aroclor 1242	12/17	350,000	1,330
Aroclor 1248	2/17	9,700	84
Aroclor 1254	3/15	3,500	185
Aroclor 1260	2/17	10,000	179
Inorganic Chemicals (mg/kg)			
antimony	4/17	38	4.5
arsenic	15/17	62	7.8
beryllium	17/17	1.3	0.49
cadmium	16/17	26	3.9
chromium	17/17	542	57
copper	17/17	8,600	431
cyanide	9/17	0.032	0.001
lead	17/17	2,810	271
mercury	16/17	13.6	0.75
nickel	17/17	116	29
selenium	3/17	2.1	0.45
silver	1/17	40	NC
zinc	17/17	1,870	338

ND = Not detected.
NC = Not calculated since chemical was detected in only one sample.

(C) = Carcinogenic PAH.
(NC) = Noncarcinogenic PAH.

chloride is a biotransformation product of several of the other unsaturated chlorinated hydrocarbons detected in soil samples. Besides vinyl chloride, no additional VOCs were selected for evaluation (other than those already selected) based on these medium depth soil results.

Twenty seven semi-volatile compounds were detected in the medium depth soil samples. All but two (acenaphthylene and benzidine) were already detected in the shallow soil samples. Acenaphthylene is a member of the noncarcinogenic PAH class which has already been selected for assessment and thus it was retained. Benzidine was only detected in one of the 17 medium depth soil samples, not detected in the shallow, deep, or very deep samples, and not detected in any other sampled media; thus it was not retained as a chemical of concern. Based on these results, no additional semi-volatiles were selected as chemicals of concern in soil.

Three pesticides (aldrin, dieldrin, methoxychlor) and PCBs were detected in the medium depth soil samples (see Table 2-4). Aldrin, dieldrin, and PCBs have already been selected as chemicals of concern. Methoxychlor was only detected in one of the 17 medium depth samples, not detected in the shallow, deep, and very deep samples, and not detected in any other sampled media; thus it was not retained for detailed evaluation in this assessment.

Table 2-4 also summarizes the results for inorganics in medium depth soil samples. Based on these data, no additional inorganic chemicals of concern were selected (other than those already selected above).

2.3.1.3 Deep (Top of Clay) Soil Samples

Table 2-5 summarizes the results of the deep soil sampling. Eighteen VOCs were detected in these samples and, with the exception of styrene, all were detected in either or both the shallow or medium depth soil samples summarized above. Styrene was detected in only one of the 17 deep soil samples, not at all in the shallow, medium, or very deep soils, and not in any other sampled media; thus it was not retained as a chemical of concern. Among the remaining VOCs, no additional chemicals (i.e., other than those noted above) were selected for detailed evaluation based on these results.

Dieldrin and PCBs, both of which have already been selected as chemicals of concern, were the only pesticides/PCBs detected in the deep soil samples. Twenty semi-volatile compounds were detected in the deep soil samples. With the exception of isophorone, all have already been detected at other depths. Isophorone was detected in three of the 17 samples and in one of seven very deep samples, but was not detected in the shallow or medium samples. Although it was infrequently detected, isophorone was observed in ground water and thus was retained as a chemical of concern in soil. Based on the deep soil samples, isophorone was the only additional semi-volatile added to the list of previously selected semi-volatile chemicals of concern.

The summary of results for inorganic chemicals in deep soil is shown in Table 2-5. Based on these data, no inorganics were added to the list of the already selected inorganics.

TABLE 2-5

SUMMARY OF CHEMICAL CONCENTRATION IN DEEP SOIL SAMPLES

Chemicals (Concentration Units)	Frequency Of Detection	Maximum Detected Concentration	Geometric Mean Concentration
Volatile Organic Compounds (ug/kg)			
Benzene	3/17	1,010	43
Chlorobenzene	2/17	115	21
Chloroform	2/17	10,300	22
1,1-Dichloroethane	2/17	234	21
1,2-Dichloroethane	4/17	6,500	36
1,2-trans-Dichloroethylene	6/17	12,200	37
Ethylbenzene	7/17	45,600	106
Methyl ethyl ketone	10/17	31,500	360
Methylene chloride	8/17	7,260	77
1,1,2,2-Tetrachloroethane	1/17	32.4	NC
1,1,1-Trichloroethane	3/17	57,600	36
m-Xylene	10/17	135,000	237
o+p-Xylenes	8/17	87,900	201
Styrene	1/17	212	NC
Tetrachloroethylene	7/17	917,000	113
Toluene	14/17	216,000	290
Trichloroethylene	7/17	363,000	45
Vinyl chloride	1/17	11,774	NC
Semi-Volatile Compounds (ug/kg)			
Acenaphthene (NC)	1/17	100	NC
Anthracene (NC)	3/17	181	52
Benzo(a)anthracene (C)	1/17	564	NC
Benzo(a)pyrene (C)	10/17	4,740	261
Benzo(b)fluoranthene (C)	1/17	576	NC
Benzo(g,h,i)perylene (NC)	1/17	227	NC
Bis(2-ethylhexyl)phthalate	13/17	3,360	2,140
Butylbenzylphthalate	3/17	4,690	380
Chrysene (C)	4/17	1,340	83
1,2-Dichlorobenzene	6/17	10,800	108
Di-n-butylphthalate	3/17	2,440	388
Di-n-octylphthalate	3/17	5,610	379
Fluoranthene (NC)	7/17	23,201	125
Fluorene (NC)	2/17	186	52
Indeno(1,2,3-cd)pyrene (C)	1/17	213	NC
Isophorone	3/17	725	83
Naphthalene (NC)	10/17	2,270	168
Phenanthrene (NC)	5/17	3,250	196
Phenol	2/17	14,400	86
Pyrene (NC)	8/17	1,840	108
Pesticides/PCBs (ug/kg)			
Dieldrin	3/17	210	4.1
Aroclor 1242	11/17	5,400	121
Aroclor 1248	3/17	2,600	22
Aroclor 1254	3/17	2,200	38
Aroclor 1260	3/17	1,000	39
Inorganic Chemicals (mg/kg)			
Antimony	2/17	29	3.6
Arsenic	10/17	18	2.8
Beryllium	17/17	0.74	0.48
Cadmium	10/17	132	0.72
Chromium	17/17	56	20.2
Copper	17/17	11,900	66.7
Lead	15/17	916	28.7
Mercury	10/17	13.6	0.16
Nickel	17/17	44	14.1
Selenium	2/17	1.3	0.28
Silver	2/17	1.2	0.55
Zinc	17/17	4,400	92

NC = Not calculated because chemical was detected in only one sample.
ND = Not detected.

(C) = Carcinogenic PAH
(NC) = Noncarcinogenic PAH

2.3.1.4 Very Deep (Within Clay Layer) Soil Samples

Table 2-6 summarizes the sampling results in very deep, within the clay layer, soils. These samples were collected from three, rather than 17, boring locations (RMW-2D, RMW-5D and RMW-7D). At each location, several samples were collected at depths down to approximately 25 feet. At these very deep soil depths, 13 VOCs, 5 semi-volatiles, 1 pesticides/PCB, and 8 inorganic chemicals were detected. Based on these results, no additional chemicals of concern were selected for evaluation (i.e., other than those already selected above).

2.3.1.5 Horizontal and Vertical Distribution of Chemicals in Soils

Trends in both vertical and horizontal distribution of chemicals detected in soil were discussed in the Dames and Moore (1990) RI. In general, VOCs, semi-volatiles (with the exception of acid extractable compounds such as 2,4-dimethylphenol), PCBs, and inorganics were present at high levels across the site with no distinct spatial pattern.

As shown in Table 11A of the Dames and Moore (1990) RI (this table compares sample results for Aroclors by depth), concentrations of PCBs generally decreased with increasing depth. The highest PCB concentrations were found in the shallow soil samples.

Dames and Moore (1990) also compare the concentrations of inorganic compounds by depth in Table 15 of their RI. As can be seen from this table, there is no clear trend in the vertical distribution of inorganics although the

A

TABLE 2-6

SUMMARY OF CHEMICAL CONCENTRATIONS
DETECTED IN VERY DEEP SOIL SAMPLES

Chemical (Concentration Units)	Frequency of Detection	Maximum Detected Concentration (ug/kg)	Geometric Mean Concentration (ug/kg)
Volatile Organic Compounds (ug/kg)			
chlorobenzene	2/16	31,523	199
chloroform	6/16	333,000	217
1,1 - dichloroethane	1/16	698	NC
1,2 - dichloroethane	3/16	59,900	206
1,2 - trans-dichloroethylene	2/16	13,820	88
ethyl benzene	2/16	69,606	221
methyl ethyl ketone	8/16	69,000	1,180
methlyene chloride	15/16	99,100	2,250
tetrachloroethylene	14/16	536,013	2,220
toluene	13/16	469,276	1,120
1,1,1 - trichloroethane	2/16	200,449	348
trichlorethylene	16/16	1,071,522	6,630
m-xylene	9/16	191,660	523
o+p - xylenes	5/16	117,053	319
Semi-Volatile Compounds (ug/kg)			
2-chlorophenol	1/7	238	NC
1,2-dichlorobenzene	2/7	465	79
isophorone	1/7	151	59
nitrobenzene	5/7	718	154
phenol	1/7	434	NC
Pesticides/PCBs (ug/kg)			
PCBs: Aroclor 1242	3/7	370	33

TABLE 2-6 (Continued)

SUMMARY OF CHEMICAL CONCENTRATIONS
DETECTED IN VERY DEEP SOIL SAMPLES

Chemical (Concentration Units)	Frequency of Detection	Maximum Detected Concentration (ug/kg)	Geometric Mean Concentration (ug/kg)
Inorganic Compounds (mg/kg)			
arsenic	5/7	5.5	1.7
beryllium	7/7	1.2	1.0
cadmium	1/7	0.28	0.15
chromium	7/7	33	28
copper	7/7	39	30
lead	6/7	17	7.2
nickel	7/7	37	3.0
zinc	7/7	87	71

NC = Not calculated since chemical was detected in only one sample.

ND = Not detected.

concentrations tend to be highest in the shallowest soil samples (i.e., 0-2 feet in depth).

Table 2-7 summarizes the geometric mean concentrations of those VOCs and semi-volatiles detected most frequently in soils at the SCP site. Geometric mean concentrations for the VOCs are highest in the saturated fill (medium depth) and within the clay (very deep). The lower concentrations of VOCs at the surface may be due, in part, to volatilization of these compounds to the ambient air. These results indicate that there is vertical migration downwards of VOCs extending down to within the clay layer. This migration is confirmed by the presence of VOCs in the till and bedrock aquifer ground water as discussed in the following section. For the semi-volatiles, geometric mean concentrations are generally higher in the fill than at the top of or within the clay. The semi-volatiles and PCBs also appear to have migrated into very deep soil; however, they are less mobile than the VOCs and few were detected in very deep soil.

2.3.2 Ground Water

Ground water samples were collected at three different depth ranges at the SCP site. Shallow well samples were collected from the water table aquifer at seven locations and deep well samples were collected from the till aquifer, which is approximately 25-50 feet below the surface, at three locations. Ground water results from both the July and December 1987 sampling rounds from these aquifers were considered valid and thus were used in this assessment.

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TABLE 2-7

COMPARISON OF GEOMETRIC MEAN ORGANIC CHEMICAL CONCENTRATIONS IN SOIL
ACROSS FOUR DEPTH RANGES AT THE SCP SITE

(All concentrations are in ug/kg) (a)

| | Soil Sampling Depth | | | |
Chemical	Shallow (0-2') (Unsaturated Fill)	Medium (5-6') (Saturated Fill)	Deep (b) (Top of Clay)	Very Deep (Within the Clay)
Volatile Organic Compounds				
1,2-trans-Dichloroethylene	9	288	37	88
Ethylbenzene	384	4,330	106	221
Methylene chloride	143	565	77	2,250
Tetrachloroethylene	934	2,760	113	2,220
Toluene	739	15,700	290	1,120
Trichloroethylene	270	856	45	6,630
Semi-Volatile Compounds				
Benzo(a)pyrene	836	649	261	ND
Bis(2-ethylhexyl)phthalate	33,600	14,400	2,140	ND
1,2-Dichlorobenzene	543	499	108	79
Fluoranthene	1,850	1,460	125	ND
Pesticides/PCBs				
Dieldrin	170	23	4	ND
PCBs (Aroclor 1242)	345	1,330	121	33

(a) Reported concentrations are geometric means from Tables 2-2, 2-4, 2-5, 2-6.

(b) The clay layer ranges from 0-18 feet in thickness across the site and begins roughly
10 feet below the site's surface.

Dames and Moore's two data sets were combined in this evaluation because of the small number of sampling locations and samples collected. To calculate the average concentrations for these data, first the geometric mean of the two samples (July and December) from each station were calculated. Then the geometric means of these means were calculated. The maximum value was, however, based on the maximum detected value in any single sample.

In addition, two ground water samples were collected from the deeper bedrock aquifer at a single on-site location at two different times by both Dames and Moore and USEPA; one sample was collected in February 1989, and the other in April 1989. The USEPA results were used in this assessment. The February sample was analyzed for both organic and inorganic chemicals, while the April sample was analyzed only for organics. A geometric mean concentration was calculated for only those organics which were detected in both samples.

2.3.2.1 Water Table Aquifer Samples

Table 2-8 summarizes the sampling results from the shallow water table aquifer. Nineteen VOCs were detected, with toluene, benzene, methylene chloride, and 1,2-trans-dichloroethylene detected most frequently. All 19 of these VOCs were also detected in soil samples with the exception of chloroethane. In addition, except for chloroethane, 1,1-dichloroethylene, and 1,1,2,2-tetrachloroethane, all of these VOCs (16) were already selected as chemicals of concern based on the soil sampling results. The same 16 VOCs selected for soil are thus also retained as chemicals of concern for ground

TABLE 2-8

CONCENTRATIONS OF CHEMICALS DETECTED IN THE WATER
TABLE AQUIFER AT THE SCP SITE

(UNFILTERED SAMPLES)

Chemical	Frequency of Detection (a)	Concentration (ug/l)	
		Geometric Mean (b)	Maximum Detected Value (b)
Volatile Compounds			
Benzene	10/14	318	7,270
Chlorobenzene	3/14	9.8	4,020
Chloroethane	1/14	NC	2,420
Chloroform	4/14	38.1	614,000
1,1-Dichloroethane	8/14	86.5	11,700
1,2-Dichloroethane	4/14	33.9	473,000
1,1-Dichloroethylene	1/14	NC	32
1,2-trans-Dichloroethylene	12/14	2,270	64,700
Ethylbenzene	6/14	35.9	3,900
Methylene Chloride	10/14	522	200,000
Methyl ethyl ketone	5/14	168	2,000,000
1,1,2,2-Tetrachloroethane	4/14	17.0	7,350
Tetrachloroethylene	3/14	16.2	24,500
Toluene	14/14	10,500	90,900
1,1,1-Trichloroethane	7/14	58.8	81,200
Trichloroethylene	8/14	365	161,000
Vinyl Chloride	9/14	106	7,290
m-Xylene	6/14	49.2	20,400
o + p-Xylenes	8/14	123	15,200
Semi-Volatile Compounds			
Total CPAHs (c)	2/14	6.8	379.5
Total NCPAHs (d)	13/14	30.7	2706.9
bis(2-Chloroethyl)ether	2/14	11.1	1,390
bis(2-Ethyl hexyl)phthalate	5/14	17.1	654
Butyl benzyl phthalate	1/14	NC	10.4
2-Chloronaphthalene	1/14	NC	18.9
2-Chlorophenol	2/14	5.9	17.8
1,2-Dichlorobenzene	12/14	34.8	192
2,4-Dichlorophenol	2/14	9.1	463
Diethyl phthalate	2/14	7.4	416
2,4-Dimethyl phenol	11/14	53.9	1,090
Dimethyl phthalate	1/14	NC	316
Di-n-butyl phthalate	2/14	7.2	318
Isophorone	5/14	26.3	8,450
Nitrobenzene	4/14	65.0	57,900
2-Nitrophenol	1/14	NC	4.73
Phenol	14/14	510	17,100
Pesticides/PCBs			
Beta-BHC	1/14	NC	0.56
Total DDT and compounds	3/14	0.09	1.7
Endrin aldehyde	2/14	0.09	15.0
Endosulfan I	1/14	NC	0.25
Endrin	1/14	NC	0.65
Total PCBs (e)	6/14	1.9	17,000
Inorganics			
Arsenic	10/14	30.7	3,100
Beryllium	4/14	1.2	4.3
Cadmium	4/14	3.5	16
Chromium	7/14	26.3	450
Copper	14/14	341	1,580
Cyanide	11/14	0.07	4.52
Lead	5/14	14.3	1,500
Mercury	10/14	0.49	4.4
Nickel	12/14	55.5	180
Zinc	14/14	92.4	2,970

(a) Frequency of detection based on 14 samples, two from each sampling station.

(b) Geometric means and maximums were calculated after the geometric mean of the two samples from each station were calculated. The listed maximum is, however, the maximum value detected in any sample.

(c) CPAHs = Carcinogenic PAHs. Those detected in groundwater were: benzo(a)pyrene, chrysene, fluoranthene and fluorene.

(d) NCPAHs = Noncarcinogenic PAHs. Those detected in groundwater were: acenaphthene, acenaphthylene, anthracene, naphthalene, phenanthrene and pyrene.

(e) Includes all Aroclors detected at site [1242].

NC = Not calculated since chemical was detected in only one sample.

water. Because chloroethane was not detected in soils, was detected in only one of the 14 water table samples and was not detected in either the till aquifer or the bedrock aquifer samples (see below), it was not retained as a chemical of concern. 1,1,2,2-Tetrachloroethane and 1,1-dichloroethylene were detected in both soil and the water table aquifer, and 1,1-dichloroethylene was also detected in the till aquifer (see below). Based on their presence in more than one environmental medium (although detected infrequently), these two VOCs are selected as chemicals of concern. Based on the VOC results from the water table aquifer, the 17 VOCs retained for evaluation in this assessment are:

chlorobenzene	1,1,1-trichloroethane
chloroform	trichloroethylene
1,1-dichloroethane	vinyl chloride
1,2-dichloroethane	benzene
1,1-dichloroethylene	ethylbenzene
methylene chloride	1,1,2,2-tetrachloroethane
tetrachloroethylene	1,2-trans-dichloroethylene
toluene	xylenes
methyl ethyl ketone	

Seventeen semi-volatiles were detected in the water table aquifer. Nine of these were already selected as chemicals of concern for soil [carcinogenic PAHs, noncarcinogenic PAHs, bis(2-ethylhexyl)phthalate, butyl benzyl phthalate, 2-chloronaphthalene, 1,2-dichlorobenzene, di-n-butyl phthalate, 2,4-dimethylphenol, and phenol]. These are thus also selected as chemicals of concern in ground water. Four chemicals detected in ground water were not detected in soil [bis(2-chloroethyl)ether, dimethyl phthalate, 2-chlorophenol, and 2-nitrophenol]. These four were detected in the water table infrequently (in one or two of the 14 samples) and were not detected in the till or bedrock

aquifer samples (see below), and thus are not retained as chemicals of

concern. Two of the remaining semi-volatiles, 2,4-dichlorophenol and

diethylphthalate, were detected in only two of the 14 water table samples,

were not detected in the till or bedrock aquifer samples, and they are

therefore not retained for evaluation in this assessment. (2,4-Dichlorophenol

was also detected in only one soil sample.) Isophorone and nitrobenzene were

detected in five and four of the 14 water table aquifer samples, respectively,

and, although they were detected very infrequently in soils (in one sample

total for 2,4-dichlorophenol and two samples total for diethylphthalate), they

are retained as chemicals of concern. Based on these results, the 11 semi-

volatiles remaining as chemicals of concern in ground water are:

carcinogenic PAHs   di-n-butyl phthalate
noncarcinogenic PAHs   2,4-dimethylphenol
bis(2-ethylhexyl)phthalate   phenol
butyl benzyl phthalate   isophorone
2-chloronaphthalene   nitrobenzene
1,2-dichlorobenzene

PCBs were detected in six of the total 14 water table samples. It should be

noted that the maximum reported PCB concentration exceeds the water solubility

of this chemical mixture. Since these samples were unfiltered prior to

analysis, all or a portion of the reported concentrations may be associated

with suspended solids that were present in the sample. To further investigate

the role of suspended materials in the water table samples, Dames and Moore

(1990) analyzed several filtered samples. It should be noted that PCBs may

adsorb to the filter and this will tend to underestimate dissolved water

concentrations. PCBs were, however, still observed in one filtered sample in

MW-3S (8,800 ug/l) above their solubility. In the other filtered samples (MW-6S, MW-7S) PCBs were not detected. Although the reason for the presence of PCBs in the one filtered sample above their solubility limit in water is not definitely known, it is possible that the presence of solvents in site soil and ground water at the site may have increased the solubility of PCBs by cosolubilization. The effect of water soluble chemicals is to exponentially increase the solubility of slightly soluble compounds such as PCBs (Morris et al. 1988, Means et al. 1989). The presence of solvents in the ground water may also enhance the potential for PCB migration in ground water. As PCBs were detected frequently in this medium (and in soil), they are retained as chemicals of concern.

Five pesticides were detected in the water table samples, but none of these were detected in any of the soil samples nor the till and bedrock aquifer samples. Thus they were not retained as chemicals of concern for ground water.

Ten inorganic compounds were detected in the water table aquifer. Although all or some portion of the inorganic chemical concentrations may be naturally occurring, no background data were available with which to determine this. Thus all of the inorganics are selected as chemicals of concern with the exception of beryllium and copper. Copper is an essential human nutrient and is not expected to pose risks to human health at the levels detected in ground water at the site. Beryllium was detected at levels below its Contract

Required Quantitation Limit (CRQL) of 5 ug/l and is not present at elevated

levels in other environmental media at the site.

Thus, the following detected inorganics are retained as chemicals of concern

based on the water table results:

arsenic	lead
cadmium	mercury
chromium	nickel
cyanide	zinc

2.3.2.2 Till Aquifer Samples

Table 2-9 summarizes the sampling results from the till aquifer. Twelve VOCs

were detected in this aquifer, and all have already been selected as chemicals

of concern based on their presence in the water table aquifer. Three semi-

volatiles and PCBs were also detected and were already selected.

Only two inorganics were detected, one of which has been selected (zinc) and

one of which was not selected because it is an essential human nutrient

(copper).

Based on the till aquifer results, no additional chemicals of concern were

selected. The till aquifer sampling data indicate that there has been

downward migration of site-related chemicals from the soil and water table

aquifer into the till aquifer, especially the migration of VOCs which are

generally most mobile in the environment.

TABLE 2-9

CONCENTRATIONS OF CHEMICALS DETECTED IN THE
TILL AQUIFER AT THE SCP SITE

(UNFILTERED SAMPLES)

Chemical	Frequency of Detection (a)	Concentration (ug/l)	
		Geometric Mean (b)	Maximum Detected Value (b)
Volatile Compounds			
Chlorobenzene	2/6	4.6	39.7
Chloroform	5/6	324	28,600
1,1-Dichloroethane	1/6	NC	27
1,2-Dichloroethane	5/6	144	9,230
1,1-Dichloroethylene	3/6	17.3	313
1,2-trans-Dichloroethylene	3/6	11.6	190
Methylene Chloride	6/6	101	1210
Tetrachloroethylene	4/6	26.7	996
Toluene	2/6	3.1	10.1
1,1,1-Trichloroethane	4/6	29.5	417
Trichloroethylene	6/6	410	16,400
Vinyl Chloride	1/6	NC	54.3
Semi-Volatile Compounds			
1,2-Dichlorobenzene	2/6	5.4	7.46
Nitrobenzene	3/6	7.2	23.3
Phenol	1/6	NC	2.16
Pesticides/PCBs			
Total PCBs (c)	1/6	NC	1.8
Inorganics			
Copper	1/6	NC	19
Zinc	5/6	29.5	57

(a) Frequency of detection based on 6 samples, two from each of the three
    sampling stations.
(b) Geometric means and maximums were calculated after the geometric mean of the two
    samples from each station were calculated. The listed maximum is, however,
    the maximum value detected in any sample.
(c) Includes all Aroclors detected at site.

NC = Not calculated because chemical was detected in only one sample.

2.3.2.3 Bedrock Aquifer Samples

Table 2-10 summarizes the sampling results from the bedrock aquifer. Ten VOCs were detected in one or both ground water samples from this aquifer. All ten VOCs have already been selected as chemicals of concern in ground water. No semi-volatiles on the Hazardous Substance List (HSL), pesticides or PCBs were detected in either bedrock aquifer sample.

Analysis of the February 1989 sample showed 11 inorganics present above their detection limits. Zinc, chromium and lead have already been selected as chemicals of concern because of their presence in the water table aquifer. Barium and vanadium were uniquely detected in the bedrock aquifer sample. Because these two chemicals were detected at levels below their contract required quantitation limits (CRQLs) and in the field blank, but not the other shallower ground water samples, they were not retained as chemicals of concern. The six remaining inorganics (aluminum, calcium, copper, magnesium, potassium, and sodium) are essential human nutrients and are not likely to be of concern to human health at the levels detected in the bedrock aquifer ground water.

Based on the sampling results from the bedrock aquifer, no additional chemicals of concern were selected. The results, however, indicate that further downward migration of previously selected VOCs has occurred from the till aquifer and overlying soils to the bedrock aquifer.

TABLE 2-10

CONCENTRATIONS OF CHEMICALS DETECTED IN THE BEDROCK AQUIFER
AT THE SCP SITE

(UNFILTERED SAMPLES)

Chemical	Frequency of Detection (a)	Concentration (ug/l)	
		Geometric Mean	Maximum Detected Value
Volatile Compounds			
Chloroform	2/2	670	830
1,2-Dichloroethane	2/2	420	460
1,1-Dichloroethylene	1/2	NC	2
1,2-trans-Dichloroethylene	1/2	NC	3
Methylene chloride	1/2	NC	21
Tetrachloroethylene	1/2	NC	2
Toluene	1/2	NC	15
1,1,1-Trichloroethane	1/2	NC	8
Trichloroethylene	2/2	240	310
Vinyl chloride	2/2	28	56
Inorganics			
Aluminum	1/1	NC	863
Barium	1/1	NC	142
Calcium	1/1	NC	209,000
Chromium	1/1	NC	27.6
Copper	1/1	NC	52.3
Lead	1/1	NC	2.6
Magnesium	1/1	NC	1,380
Potassium	1/1	NC	3,100
Sodium	1/1	NC	60,500
Vanadium	1/1	NC	7
Zinc	1/1	NC	7.8

(a) Frequency of detection based on two samples for organics and one sample for inorganics. The samples were taken from a single monitoring well on two separate dates.

NC = Not calculated since chemical was detected in only one sample.

2.3.3 Surface Water

Surface water samples were collected at four locations in Peach Island Creek:
100 feet upstream of the site, adjacent to the site, 100 feet downstream of
the site, and also roughly half a mile downstream of the site at the
confluence of Peach Island Creek and Berry's Creek.

The surface water sampling data are shown in Table 2-11. A total of ten (10)
VOCs (all of which are hazardous substances) were detected in the water column
at the stations located adjacent to and/or immediately downstream from the
site. The instream VOC data reveal that the concentrations of almost all of
the VOCs detected are much higher in samples which were collected adjacent to
or immediately downstream from the site than in the other two sample
locations. The instream VOC data specifically reveal the following:

    1) The concentrations of nine of the ten VOCs listed in Table 2-11 are
        greater in the samples taken at or immediately downstream from the
        site than the levels detected upstream from the site. All nine of
        these VOCs exist in soil and/or ground water at the site.

    2) Eight VOCs were below detectable levels upstream from the site. The
        instream data collected adjacent to and/or immediately downstream
        from the site revealed much higher levels for all eight of these
        VOCs. All eight of these VOCs exist in the soil and/or ground water
        at the site.

The VOCs detected in the water column (and sediment) in Peach Island Creek
adjacent to and immediately downstream from the site are not naturally
occurring. All of the VOCs detected in the water column in Peach Island Creek
have in fact, been detected at and still exist in the soil and/or ground water

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TABLE 2-11

CHEMICAL CONCENTRATIONS IN SURFACE WATER SAMPLES
AT PEACH ISLAND CREEK

(All concentrations in ug/liter)

Chemical	100 Feet Upstream (Loc. 4)	Adjacent to site (Loc. 3)	100 Feet Downstream (Loc. 2)	Confluence with Berry's Creek (Loc. 1)
Volatile Organic Compounds				
Chlorobenzene	ND	8.34	12.20	ND
Chloroform	ND	3.58	3.56	ND
1,2-trans-Dichloroethylene	ND	35.20	33.30	3.91
Methyl ethyl ketone	75	45.40	49.20	ND
Methylene chloride	4.63	6.12	12.90	14.90
1,1,1-Trichloroethane	ND	6.32	5.54	ND
Toluene	ND	20.60	48.10	ND
Trichlorethylene	ND	3.83	ND	ND
m-Xylene	ND	ND	10.70	ND
o+p-Xylenes	ND	ND	10.00	ND
Inorganic Chemicals				
Chromium	56	ND	28	ND
Copper	100	29	27	12
Mercury	4.8	0.96	1.1	2.1
Nickel	57	33	27	ND
Zinc	370	160	150	87

ND = Not detected.

at the site. The pattern of VOC distribution in the water column in Peach Island Creek, as noted above, clearly indicates that VOCs are being and have been released from the site into Peach Island Creek. The probable routes of migration are via direct surface runoff and ground water discharge from the shallow aquifer at the site. Because of this pattern of detection of VOCs in three media (site ground water, site soils and surface water), all ten (10) of the VOCs detected in the water column near the site are retained as chemicals of concern for Peach Island Creek.

Five inorganic compounds were detected in surface water without any distinct pattern. All were retained as chemicals of concern except copper. Copper is an essential human nutrient and is only toxic to humans at very high levels (higher than could be associated with surface water contact at the site), although as will be seen in Section 6, it will be selected as a chemical of concern for its potential to cause adverse ecological effects.

2.3.4 Sediments

Shallow (0-6 inches) and deep (12-18 inches) sediment samples were taken at the same locations in Peach Island Creek as were surface water: 100 feet upstream, adjacent to the site, 100 feet downstream, and at the confluence of Peach Island Creek and Berry's Creek. Tables 2-12 and 2-13 summarize these results for shallow and deep sediments, respectively.

TABLE 2-12

SUMMARY OF CHEMICAL CONCENTRATIONS
IN SHALLOW SEDIMENTS (0-6 INCHES)

Chemical	Concentration			
	100 Feet Upstream (Loc. 4)	Adjacent to Site (Loc. 3)	100 Feet Downstream of Site (Loc. 2)	Confluence with Berry's Creek (Loc. 1)
Volatile Organic Compounds (ug/kg)				
Benzene	ND	ND	ND	82.5
Chlorobenzene	3,990	ND	17,100	200
Chloroform	ND	ND	3,690	ND
Ethylbenzene	4,610	39,000	35,100	ND
Methyl ethyl ketone	ND	ND	18,300	65.2
Methylene chloride	ND	ND	ND	42.3
m-Xylene	13,300	1,060,000	72,000	168
o+p-Xylenes	11,000	647,000	74,200	467
Tetrachloroethylene	ND	953,000	ND	ND
Toluene	41,500	2,970,000	322,000	ND
1,1,1-Trichloroethane	ND	222,000	ND	ND
Trichlorethylene	ND	9,950,000	ND	ND
Pesticides/PCBs (ug/kg)				
Dieldrin	ND	11,000	ND	ND
PCBs:				
Arochlor 1242	21,000	55,000	35,000	ND
Arochlor 1248	ND	ND	ND	19,000
Arochlor 1254	ND	ND	ND	5,200
Arochlor 1260	10,000	ND	6,000	ND
Semi-Volatiles (ug/kg)				
1,2,4-Trichlorobenzene	525	ND	ND	ND
1,2-Dichlorobenzene	1,850	3,670	424	ND
2-Chloronaphthalene	ND	ND	115	ND
Acenaphthene	ND	ND	166	ND
Benzo(a)pyrene	ND	ND	148	ND
Bis(2-ethylhexyl)phthlate	108,000	32,600	32,000	2,920
Butyl benzyl phthalate	ND	ND	736	ND
Chrysene	ND	ND	332	ND
Di-n-octylphthalate	ND	ND	600	ND

TABLE 2-12 (continued)

SUMMARY OF CHEMICAL CONCENTRATIONS
IN SHALLOW SEDIMENTS (0-6 INCHES)

Chemical	Concentration			
	100 Feet Upstream (Loc. 4)	Adjacent to Site (Loc. 3)	100 Feet Downstream of Site (Loc. 2)	Confluence with Berry's Creek (Loc. 1)
Semi-Volatiles (ug/kg) (Cont'd)				
Di-n-butylphthalate	2,350	ND	ND	ND
Fluoranthene	928	ND	374	ND
Fluorene	536	ND	202	ND
Naphthalene	1,330	816	1,230	ND
Phenanthrene	1,820	ND	712	ND
Pyrene	916	ND	339	ND
2,4-Dimethylphenol	1,360	ND	ND	ND
Phenol	24,900	10,200	ND	ND
Inorganics (mg/kg)				
Arsenic	37	ND	ND	34
Beryllium	2.4	1	0.39	0.7
Cadmium	84	43	12	32
Chromium	819	345	156	1,060
Copper	9,510	2,000	1,240	861
Cyanide, total	0.12	0.21	0.001	0.005
Lead	320	520	340	360
Mercury	41	25	0.34	139
Nickel	467	110	96	100
Selenium	ND	ND	ND	0.89
Silver	2.4	2.7	ND	8.6
Thallium	1.0	ND	ND	1.1
Zinc	3,110	2,320	411	2,880

ND = Not detected.

TABLE 2-13

SUMMARY OF CHEMICALS CONCENTRATIONS IN SAMPLES OF DEEP SEDIMENTS

	Concentration			
Chemical	100 Feet Upstream (Location 4)	Adjacent to Site (Location 3)	100 Feet Downstream of Site (Location 2)	Confluence With Berry's Creek (Location 1)
VOLATILE ORGANIC COMPOUNDS (ug/kg)				
1,1,1-Trichloroethane	ND	75,500	ND	ND
1,2-Dichloroethane	1,960	ND	ND	ND
1,2-trans-Dichloroethylene	1,160	ND	ND	ND
Benzene	1,990	ND	5,785	33.4
Chlorobenzene	4,930	ND	ND	47.3
Chloroethane	ND	ND	2,127	ND
Chloroform	3,790	ND	ND	ND
Ethylbenzene	7,420	174,000	ND	29.7
Methyl ethyl ketone	31,900	ND	ND	ND
Methylene chloride	3,690	ND	ND	ND
Tetrachloroethylene	ND	304,000	ND	ND
Toluene	74,500	1,700,000	726	ND
Trichloroethylene	1,890	3,260,000	ND	ND
m-Xylene	17,200	486,000	5,796	93.8
o+p-Xylenes	16,000	348,000	9,481	141
BASE NEUTRALS (ug/kg)				
1,2,4-Trichlorobenzene	177	2,330	ND	ND
1,2-Dichlorobenzene	445	261,000	852	ND
Bis(2-ethylhexyl)phthalate	32,600	240,000	95,651	5,700
Butyl benzyl phthalate	ND	9,700	ND	ND
Chrysene	ND	ND	1,010	ND
Dibenzo(a,h)anthracene	ND	ND	870	ND
Di-n-butyl phthalate	884	24,800	2,791	ND
Di-n-octyl phthalate	ND	12,200	938	ND
Fluoranthene	381	ND	1,465	534
Naphthalene	379	20,300	1,014	ND
Phenanthrene	556	ND	2,569	ND
Phenol	6,560	44,700	ND	ND
Pyrene	343	ND	1,254	ND
PESTICIDES/PCBs				
PCBs:				
Aroclor 1242	8,880	770,000	21,675	ND
Aroclor 1248	ND	ND	ND	42,000
Aroclor 1254	ND	ND	ND	5,500
Aroclor 1260	2,800	ND	11,099	ND
INORGANICS (mg/kg)				
Arsenic	15	22	7.4	31
Beryllium	1.4	2	0.62	0.63
Cadmium	29	74	30	28
Chromium	257	504	258	1.170
Copper	2,230	2,590	1,213	835
Lead	96	230	232	370
Cyanides, total	0.02	0.01	0.014	0.002
Mercury	18	41	1.93	1,390
Nickel	203	413	228	140
Silver	ND	ND	ND	7.6
Thallium	ND	1.1	ND	1.2
Zinc	1,060	2,540	945	3,680

A total of 15 VOCs were detected in the deep sediments near the site, all of which are hazardous substances under CERCLA. Twelve (12) of these 15 were also detected in the shallow sediments near the site.

The shallow sediment data indicate that the concentrations of almost all the VOCs detected are much higher in samples which were collected adjacent to and/or immediately downstream from the site, as compared to samples collected upstream from the site. The shallow sediment VOC data specifically reveal the following:

1) The concentrations of 10 of the 12 VOCs detected are greater adjacent to and immediately downstream from the site than the levels detected upstream from the site. (The two other VOCs were not detected adjacent to or immediately downstream from the site). All 10 of these VOCs exist in site soil and/or ground water and all are hazardous substances.

2) Seven VOCs were below detectable levels upstream from the site. The shallow sediment data collected adjacent to or immediately downstream of the site showed much higher concentrations for five of these (the other two were only detected at the confluence with Berry's Creek). All of these VOCs exist in the soil and/or ground water at the site.

3) The five VOCs which were detected upstream from the site were also present at much higher levels (by up to two orders of magnitude) adjacent to and/or immediately downstream from the site. All of these VOCs exist in soil and/or ground water at the site.

The pattern of VOC distribution in the shallow sediment, as indicated above, clearly indicates that many VOCs which are hazardous substances are being and have been released from the site into Peach Island Creek. The probable migration routes are via direct surface runoff and ground water discharge from the shallow water table aquifer at the site.

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It should also be noted that some of the VOCs detected at the site and which are present in the shallow and deep sediments in the creek were also detected in the water column in Peach Island Creek. This indicates that the sediments may also be releasing these VOCs into the water column, in addition to the other migration routes mentioned above.

With the exception of chloroethane, all of the VOCs detected in sediments were already selected as chemicals of concern based on their presence in soil and ground water at the site. In addition, all but tetrachloroethylene, ethylbenzene, and benzene, were also detected in surface water samples. Chloroethane was detected in one deep sediment sample 100 feet downstream of the site, in one ground water sample, and not at all in on-site soil. Based on its infrequent detection, it is not retained as a chemical of concern. The presence and distribution of the rest of the VOCs across several environmental media at the site indicate that site-related VOCs are being transported into Peach Island Creek sediments as well as into surface water from the site.

Data collected from the shallow and deep sediments also indicate that PCBs (Arochlors 1242 and 1260) exist in the sediment near the site. These are the same Arochlors which exist in soils and ground water at the site. Furthermore, Arochlor 1242, which is by far the most prevalent PCB discovered in soil and ground water at the site, is also the dominant PCB discovered in the creek sediments. Dieldrin was also discovered in the shallow sediment in the creek, only at the station located adjacent to the site. Dieldrin also

exists in the soil and ground water at the site. The PCB and dieldrin sediment data indicate that PCBs and dieldrin have likely been released from the site and are presently retained in the sediment in Peach Island Creek near the site. PCBs and dieldrin have already been selected as chemicals of concern based on their presence in other environmental media, and thus they are also considered chemicals of concern for sediments.

Seventeen semi-volatiles were detected in shallow sediments and 13 were detected in deep sediments. With the exception of 1,2,4-trichlorobenzene, all these semi-volatiles were detected in site soil samples, all have already been selected as chemicals of concern based on the soil results, and all are also retained as chemicals of concern for sediments. 1,2,4-Trichlorobenzene was detected in one shallow sediment sample (upstream only), two deep sediment samples (upstream and adjacent to site), in a total of four soil samples, and not at all in ground water. For the purposes of this assessment (since 12 semi-volatiles have already been selected) this chemical was not retained as a chemical of concern.

All of the semi-volatiles detected in deep sediments adjacent to and/or immediately downstream of the site were present at much higher concentrations at these two locations than in the upstream sample. There is, however, no obvious trend in the shallow sediment semi-volatile concentrations as for VOCs in these sediments.

Thirteen inorganics were detected in both shallow and deep sediments. There is no clear trend in the spatial distribution of the inorganics. The highest concentrations in shallow sediments were, for example, observed at the upstream or furthest downstream stations for all inorganics except total cyanide and lead. As mentioned earlier, inorganic compounds are naturally occurring in the environment. Background data on inorganics in sediment were not available; however, for this assessment the regional background soil data were compared with the sediment results to select chemicals of concern. The approach used for soil was also used for sediments, that is if a chemical's sediment levels were within the background soil range, it was not selected as a chemical of concern. Based on this comparison, beryllium was not retained as a chemical of concern. Although lead and nickel also were present at levels within the regional soil range, they were already selected as chemicals of concern (in soil, ground water, and surface water). Thus they were retained as chemicals of concern.

Chromium and zinc exceeded the background range, but only at the furthest downstream station. Dames and Moore (1990) concluded, based on their site investigation and a review of aerial photographs, that the results at this sampling station may be influenced more by sediments from Berry's Creek than from Peach Island Creek. Chromium and zinc were, however, already selected as chemicals of concern based on their presence in ground water and surface water, and thus they are retained as chemicals of concern in sediments. Copper was not selected because it is an essential human nutrient unlikely to

be of concern to public health at the levels detected. The nine inorganics

remaining as chemicals of concern in sediments are:

arsenic   mercury
cadmium   selenium
chromium   silver
cyanide   thallium
                   zinc

2.3.5 Summary of Chemicals of Concern

Based on this review of the Dames and Moore (1990) site data, the 1989 USEPA

bedrock ground water data and use of the selection criteria described above

(Section 2.3), a set of chemicals of concern has been selected for detailed

analysis in this baseline risk assessment (see Table 2-14). Several points

should be noted regarding these chemicals of concern, although not all of the

following information provided the basis for actually selecting the chemicals:

(1)   All are hazardous substances under CERCLA.

(2)   None of the VOCs, pesticides, PCBs and semi-volatile compounds listed
       are naturally occurring but yet many of these chemicals exist at
       grossly elevated levels at the site.

(3)   Some of the chemicals listed are possible human carcinogens (e.g.,
       butyl benzyl phthalate, 1,1-dichloroethylene, isophorone, 1,1,2,2-
       tetrachloroethane) [see Section 4].

(4)   Many are probable human carcinogens (e.g., PCBs, chloroform, 1,2-
       dichloroethane, methylene chloride, trichloroethylene, cadmium
       [inhalation only]). [See Section 4]

(5)   Some are known carcinogens in humans (e.g., vinyl chloride, arsenic,
       benzene) [see Section 4].

TABLE 2-14

SUMMARY OF SELECTED CHEMICALS OF CONCERN AT THE SCP SITE

CHEMICAL[a]	MEDIUM			
	SOIL	GROUND WATER	SURFACE WATER	SEDIMENTS
Volatile Organic Compounds				
benzene	X	X	ND	X
chlorobenzene	X	X	X	X
chloroform	X	X	X	X
1,1-dichloroethane	X	X	X	ND
1,2-dichloroethane	X	X	X	X
1,1-dichloroethylene	X	X	ND	ND
1,2-trans-dichloroethylene	X	X	X	X
ethylbenzene	X	X	ND	X
methyl ethyl ketone	X	X	X	X
methlyene chloride	X	X	X	X
1,1,2,2-tetrachloroethane	X	X	ND	ND
tetrachloroethylene	X	X	ND	X
toluene	X	X	X	X
1,1,1-trichloroethane	X	X	X	X
trichlorethylene	X	X	X	X
vinyl chloride	X	X	ND	ND
xylenes (o+p,m)	X	X	X	X
Pesticides/PCBs				
aldrin	X	ND	ND	ND
dieldrin	X	ND	ND	X
polychlorinated biphenyls (PCBs)	X	X	ND	X
Semi-Volatile Compounds				
bis-2(ethylhexyl)phthalate	X	X	ND	X
butyl benzyl phthalate	X	X	ND	X
2-chloronaphthalene	X	X	ND	X
1,2-dichlorobenzene	X	X	ND	X
2,4-dimethylphenol	X	X	ND	X
di-n-butyl phthalate	X	X	ND	X
di-n-octyl phthalate	X	X	ND	X
isophorone	X	X	ND	ND
nitrobenzene	X	X	ND	ND
PAHs, carcinogenic	X	X	ND	X
PAHs, noncarcinogenic	X	X	ND	X
phenol	X	X	ND	X

TABLE 2-14 (Continued)

SUMMARY OF SELECTED CHEMICALS OF CONCERN AT THE SCP SITE

| | MEDIUM | | | |
CHEMICAL[a]	SOIL	GROUND WATER	SURFACE WATER	SEDIMENTS
Inorganic				
antimony	X	ND	ND	ND
arsenic	X	X	ND	X
cadmium	X	X	ND	X
chromium	X	X	X	X
cyanide	X	X	ND	X
lead	X	X	ND	X
mercury	X	X	X	X
nickel	X	X	X	X
selenium	X	ND	ND	- -
silver	X	ND	ND	X
zinc	X	X	X	X

[a] Only chemicals selected in one or more medium are included.

PAH = Polycyclic aromatic hydrocarbon.
ND = Not detected.
- - = Not selected (see text for discussion).

(6)   Many exist in the water table aquifer at the site at levels which far exceed (often by orders of magnitude) the Maximum Contaminant Levels (MCLs) established for such substances pursuant to the Federal Safe Drinking Water Act (e.g., benzene and vinyl chloride (known human carcinogens), and chlorobenzene, chloroform, 1,2-dichloroethane, 1,2-trans-dichloroethylene, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane) [see Section 5.1].

(7)   Many exist in the till aquifer at the site at levels which exceed (often by orders of magnitude) the MCLs which were established for substances pursuant to the Federal Safe Drinking Water Act. (e.g., 1,2-dichloroethane, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane, chlorobenzene, and vinyl chloride [a known human carcinogen]) [see Section 5.1].

(8)   Some chemicals exist in the bedrock aquifer at levels which exceed (in some cases by more than an order of magnitude) MCLs (e.g., 1,2-dichloroethane, trichloroethylene) [see Section 5.1].

(9)   Some chemicals exist in the shallow water table aquifer at the site at levels which far exceed the Class GW-2 drinking water standards set by the State of New Jersey for such chemicals in this aquifer (e.g., PCBs) [see Section 5.1].

(10)   Many of the selected chemicals existing at the site are known to cause acute and/or chronic health effects in humans (other than carcinogenic effects) if ingested, inhaled, or dermally contacted in sufficient quantities [see Section 4].

(11)   Many of the selected chemicals which exist at the site and which were also detected in the sediment of Peach Island Creek are known to be acutely and/or chronically toxic to aquatic organisms [see Section 6].

(12)   Some of the selected chemicals which exist at the site and which were also detected in the sediment of Peach Island Creek are known to bioaccumulate and biomagnify in certain aquatic species (e.g., PCBs) [see Section 6].

(13)   Many of the selected chemicals are highly mobile in ground water (as indicated in Table 3 in the Dames and Moore (1990) RI [see Section 3.1].

(14)   Almost all of the selected VOCs (14 of 17) which exist in the soil and ground water at the site were also detected in either the water column and/or sediment in Peach Island Creek.

(15)   Almost all of the selected semi-volatile compounds (10 of 12) which exist in the soil and ground water at the site were also detected in the sediment in Peach Island Creek.

(16)   Some of the selected chemicals which exist at the site were also detected in Peach Island Creek at levels which exceed the applicable Class SE standards for that Creek (e.g., copper, mercury, nickel, zinc) [see Section 6].

(17)   The site is presently uncapped and open to the atmosphere. Many of the chemicals discovered at the site are known to be capable of volatilizing into the atmosphere and thereby migrating away from the site in ambient air [see Section 3.1].

(18)   The site receives roughly seven million gallons per year of precipitation, some of which undoubtedly flows off the site in the form of surface runoff into Peach Island Creek. Some precipitation will also infiltrate into the shallow water table aquifer [see Section 2.2]. No controls or catchment structures exist to prevent this migration at present. Therefore, many of the hazardous substances listed in Table 2-14 may migrate into this creek, especially during and shortly after storm events with consequential unknown impacts on aquatic biota.

It should be noted that because the SCP site is located in a heavily industrialized area, a portion of the detected levels of some of the selected chemicals of concern on site may also be due to background sources. For example, PAHs are virtually ubiquitous in urban and industrial areas. They are emitted by combustion sources such as the burning of coal, oil, refuse, and diesel fuel. Other sources of PAHs include vehicle tires, leaching from coal storage piles, creosote-treated lumber, or asphalt surfaces. Chlorinated pesticides such as dieldrin, which has been widely used in the U.S., are sometimes found at trace levels in both industrial and non-industrial areas. However, the levels of many of the organic hazardous substances at the site are grossly elevated in the soil and ground water; therefore, background contamination is likely to represent a minor component of the measured contaminant concentrations at the site. Concentrations of inorganic chemicals at the site may also reflect inputs from human activities as well as natural

(background) abundances. However, to determine the potential contribution of possible background sources of inorganic chemicals to on-site chemical levels, more detailed sampling data from the local area would be needed.

A

3.0 EXPOSURE ASSESSMENT

This section addresses the potential pathways by which human populations could

be exposed to contaminants at, or originating from, the SCP site. In

identifying potential pathways of exposure, both current and possible future

site and surrounding land use conditions are considered.

An exposure pathway is composed of the following four elements (USEPA 1989a):

    (1)   a source and mechanism of chemical release to the environment;

    (2)   an environmental transport medium (e.g., air) for the released
            chemical,and/or mechanism of transfer of the chemical from one
            medium to another (e.g., deposition of particles onto soil);

    (3)   a point of potential contact of humans or biota with the
            contaminated medium (the exposure point); and

    (4)   an exposure route (e.g., inhalation) at the exposure point.

An exposure pathway is considered to be "complete" if all of these elements

are present.

In this assessment, we follow a general approach to exposure assessment which

has been described in general terms by the USEPA (1986a, 1989a). First, the

potential migration characteristics of the selected chemicals of concern are

discussed. Second, the potential exposure pathways most likely to be

important at the SCP site are extracted from a list of all possible pathways.

This screening process focuses the assessment on the most important potential

exposure pathways and is justified by experience, which shows that in most

circumstances exposure to a few chemicals via a few exposure pathways

dominates estimates of potential risks. For each selected exposure pathway, the concentrations of the chemicals of concern are then estimated at the potential points of exposure (i.e., exposure point concentrations), such as nearby residences, and nearby industrial facilities.

3.1 MIGRATION OF SITE CONTAMINANTS

An important step in identifying exposure pathways is to consider the mechanisms by which the chemicals of concern at the site may migrate in the environment. The migration of chemicals that have been or may be released from the site are influenced by environmental factors, such as the hydrogeological characteristics of the site and surrounding area, the characteristics of the source area, and the physical/chemical characteristics of the chemicals themselves. A brief discussion of the mechanisms by which the selected chemicals of concern have migrated in the past and may continue to migrate in the future follows.

3.1.1 <u>Chemical and Physical Properties</u>

As discussed in Section 2.3, a wide variety of volatile organic compounds (VOCs), PCBs, certain pesticides (aldrin and dieldrin), semi-volatile compounds, and inorganics have been observed at the site. The potential for exposures to occur to these chemicals will be influenced by their distribution and their mobility and/or persistence in the environment.

The behavior of chemicals in the environment can be roughly estimated by examining several chemical and physical properties of the selected chemicals of concern. Table 3 of the Dames and Moore (1990) RI, reproduced in Appendix A of this report, provides information on the vapor pressure, water solubility, octanol:water partition coefficient (K_{ow}), and organic carbon partition coefficient (K_{oc}) of each organic chemical detected at the site.

Water solubility (S), one of the listed parameters in Appendix A, is an important property affecting environmental fate; highly soluble chemicals can be leached from soils by infiltrating precipitation and are generally mobile in various media. Water solubilities range from less than 1 mg/l to totally miscible with water, with most common organic chemicals falling between 1 mg/l and 10,000 mg/l (Lyman et al. 1982).

Volatilization of a compound from an environmental medium will depend on its vapor pressure, water solubility, and diffusion coefficient. Highly water soluble compounds generally volatilize to a lesser extent than compounds having low water solubility. If a highly water soluble compound has a high vapor pressure, it will volatilize to a greater extent than one with a low vapor pressure. Vapor pressure (VP), a relative indicator of the volatility of chemicals in their pure state, ranges from 0.001 to 760 mmHg for liquids, with solids ranging down to less than 10^{-10} mmHg. The Henry's law constant (H), which combines vapor pressure with solubility (i.e., H = VP/S), is more appropriate than vapor pressure alone for estimating releases to air from water. Compounds with Henry's law constants in the range of 10^{-3} atm-m^3/mol

and larger can be expected to volatilize readily from water; those with values

ranging from 10^{-5} to 10^{-3} atm-m^3/mol are associated with significant

volatilization but to a lesser extent, while compounds with values less than

10^{-5} atm-m^3/mol volatilize from water only to a limited extent (Lyman et al.

1982).

The octanol:water partition coefficient (K_{ow}) is an important environmental

parameter. This equals the ratio of a chemical's concentration in an octanol

phase to its concentration in an aqueous phase. The K_{ow} is often used to

estimate the extent to which a chemical will partition from water into lipid

parts of organisms, for example fish and animal fat. The chemicals of concern

for the SCP site have K_{ow} values (based on Dames and Moore's Table 3) ranging

from 1.8 to 1.6x10^9.

Similar to the K_{ow}, the organic carbon partition coefficient, K_{oc} [referred to

as the soil/sediment adsorption coefficient in the Dames and Moore (1990)

Table 3], reflects the propensity of a compound to sorb to organic matter

found in soil and sediment. Typical values of K_{oc} may range from 1 to 10^7

with higher values indicating greater sorption potential. The K_{oc} values for

the chemicals of concern for this risk assessment (based on Dames and Moore's

Table 3) range from 3.9 to 4.1x10^8.

The chemicals of concern being evaluated in this assessment can be classified

into general categories according to their similarity in chemical structure

and/or physicochemical properties (i.e., factors that would influence mobility

$\mathcal{A}$

in the environment as discussed below). The chemical categories and chemicals

of concern within each category are listed below.

Substituted simple aromatics: benzene, chlorobenzene, 1,2-
dichlorobenzene, nitrobenzene, toluene, xylenes, ethylbenzene

Chlorinated aliphatics: chloroform, methylene chloride,
tetrachloroethylene, 1,1-dichloroethane, 1,2-dichloroethane, 1,2-trans-
dichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane,
trichloroethylene, 1,1,2,2-tetrachloroethane, vinyl chloride

Ketones: methyl ethyl ketone, isophorone

Chlorinated pesticides: aldrin, dieldrin

Phenols: phenol, 2,4-dimethylphenol

Inorganics (potentially behaving as cations in water): antimony,
beryllium, cadmium, trivalent chromium, copper, lead, mercury, nickel,
silver, and zinc

Inorganics (potentially behaving as anions in water): arsenic,
hexavalent chromium, cyanide, antimony, selenium

Polynuclear aromatic hydrocarbons (PAHs): acenaphthene, acenaphthylene,
anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene,
benzo(g,h,i)perylene, chrysene, fluoranthene, fluorene, indeno(1,2,3-
cd)pyrene, naphthalene, phenanthrene, pyrene

Phthalate esters: bis(2-ethylhexyl)phthalate, butyl benzyl phthalate,
di-n-butyl phthalate, di-n-octyl phthalate

PCBs: Aroclors 1242, 1248, 1254, 1260

Miscellaneous: 2-chloronaphthalene

3.1.2 Mechanisms of Migration and Transformation

Analysis of samples collected from soil, ground water, surface water, and

sediment at the SCP site indicate that chemicals of concern together with

their transformation products are present in several or all of these media.

In this section, the mechanisms of migration and transformation of site-related chemicals (i.e., the chemicals of concern) will be discussed from a conceptual standpoint.

The potential migration routes for chemicals at the SCP site include: migration from soil downward into ground water, migration from shallower ground water (i.e., water table and till aquifers) to deeper ground water (i.e., bedrock aquifer), surface soil runoff into Peach Island Creek, migration from ground water into Peach Island Creek, lateral migration of on-site ground water to off site areas, migration into air by volatilization or by suspension of soil, biotransformation of chemicals into more toxic and/or more mobile breakdown products, migration to Peach Island Creek surface water and sediments both downstream and upstream (due to the tidal nature of the creek), and bioaccumulation of chemicals in biota impacted by the site.

3.1.2.1 Migration/Mobility in Soil and Ground Water

Tables 2-2, 2-4, 2-5, 2-6, and 2-7 summarize data on the prevalence and concentrations of chemicals in soil at the SCP site. All of the chemical classes noted above in Section 3.1.1 are represented. Some of the compounds within these classes will migrate in soil due to the presence of infiltrating precipitation and the downward hydraulic gradient between the water table and till aquifers (Dames and Moore 1990). Organic chemicals with high solubilities and low organic carbon partition coefficients (K_{oc}s) are particularly susceptible to this phenomenon. The substituted simple

aromatics, chlorinated aliphatics and phenols have aqueous solubilities in the hundreds of milligrams per liter range and K_{oc}s of approximately 5,000 or less and are, therefore, expected to be more mobile than the other chemicals of concern. The propensity of these chemicals to be mobile in soils is observed at the SCP site, where the substituted simple aromatics and chlorinated aliphatics have been observed in all soil depths sampled and in the water table, till, and bedrock aquifers. Recent water quality data collected from the bedrock aquifer confirms that migration into the bedrock aquifer, which is used as a public water supply, has occurred.

In general, the chlorinated pesticides, PAHs, PCBs, and phthalate esters detected in soils are not as mobile as the chemicals mentioned above. This is evidenced by the generally higher K_{oc}s of these chemicals (approximately 5,000 and higher) and their relatively low water solubilities (roughly 15 mg/l and less). Although these chemicals are not considered to be very mobile in soil, many are present at all the soil depths sampled at the SCP site. This is particularly true for PCBs and many of the PAHs. It should be noted that a few of the PAHs are more mobile in soils (e.g., naphthalene and acenaphthylene) than the rest of the PAH class. The presence of the less mobile PAHs and PCBs at varying soil depths indicates that downward transport is occurring, although to a lesser extent than the simple substituted aromatics and chlorinated aliphatics. This is not surprising since compounds with low water solubilities and high K_{oc} values, once in the ground water, are transported in the direction of the ground water flow but at a slower rate than the ground water. This is because compounds moving with the ground water

flow tend to partition or divide themselves between the mobile water or aqueous phase and the stationary soil particles that are in contact with ground water. The overall effect of this sorption process is a retardation of the rate of a compound's transport. The absence of these less mobile compounds from the bedrock aquifer samples, in contrast to the presence of many VOCs, may result from their lower mobilities in the environment.

As discussed in Dames and Moore (1990) and earlier in this report, PCBs were present in unfiltered samples collected from both the water table and till aquifers, and in filtered water table aquifer samples at levels exceeding their solubility. This may result from the presence of organic solvents which may enhance the solubility (and thus mobility) of PCBs in soil (Nkedi-Kizza et al. 1985). The halogenated aliphatic and monocyclic aromatic chemicals of concern have high solubilities and low K_{oc}s which indicates that they are very mobile in ground water.

The migration of inorganic chemicals through soil to ground water is also influenced by soil characteristics and water movement. Soil parameters specific to inorganics that must be considered are cation and anion exchange capacities (that is the interaction between positively and negatively charged ions), fraction of organic matter, pH, oxidation-reduction potential, and porosity. In general, inorganic chemicals with a positive charge (cations) will be retarded by clays exhibiting an overall negative charge, and anions such as chromium (as chromate) or arsenic (as arsenate) will be more mobile in such an environment.

The potential for inorganic chemicals to migrate as a result of infiltration is supported by the EP Toxicity test results from 12 soil grab samples taken at the SCP site in January 1989. The results indicated that concentrations leached from soil samples exceeded RCRA EP Toxicity concentrations in six or more of the samples for arsenic, chromium, and lead. The EP Toxicity limits were also exceeded for barium (one sample), cadmium and selenium (3 samples each), and mercury (4 samples). Each of the 12 samples contained at least one inorganic chemical which leached in excess of the EP toxicity limit.

3.1.2.2 Migration/Mobility in Surface Water and Sediment

Peach Island Creek flows along the northeastern edge of the site and is tidal. Many of the chlorinated aliphatics and substituted simple aromatics detected in Peach Island Creek adjacent to the site were also detected in the water table aquifer and in soils at the site (e.g., 1,1,1-trichloroethane, 1,2-trans-dichloroethylene). The surface of the water table aquifer is approximately five feet higher than the surface elevation of the creek. Horizontal flow in this aquifer has been reported to be radially away from the site, and, as Dames and Moore (1990) concluded, it appears that the more mobile compounds in the water table aquifer are entering Peach Island Creek. Similarly, several of the more mobile chlorinated aliphatics and substituted aromatics detected in Peach Island Creek are also present in the till aquifer.

The till aquifer appears to flow in a northwesterly direction towards Gotham Parkway and somewhat towards Peach Island Creek (see Figures 39-46 in the Dames and Moore RI). Again, the data suggest that the mobile compounds detected in the till aquifer may be entering Peach Island Creek.

The till aquifer is tidally influenced although in a different pattern than the creek. The similarity of compounds detected in Peach Island Creek, and ground water and soil at the SCP site and the high likelihood that contaminants migrate via surface runoff and ground water discharge strongly suggest migration of contaminants from the site to the creek.

Likewise, there are clearly similarities between the types of chemicals detected in sediments and those detected on site in ground water and soil. Several chlorinated aliphatics and substituted aromatics were detected at their highest levels in sediments adjacent to the site and also at elevated levels on site (e.g., 1,1,1-trichloroethane, ethylbenzene, xylenes, toluene, trichloroethylene). In addition, some of the less mobile PAHs, phthalate esters, PCBs and dieldrin were also detected at their highest levels adjacent to the site. These data strongly suggest that site-related contaminants are migrating into Peach Island Creek. One mechanism for migration may be surface runoff from the site into the creek.

The distribution of site-related contaminants in the water column and sediment in Peach Island Creek also strongly supports the conclusion that many of these contaminants are being released into the creek from the site.

3.1.2.3 Migration Into Air

Chemicals present at the SCP site may migrate into the air in two ways, by volatilization or by suspension of soil (i.e., generation of fugitive dusts). The mechanisms of volatilization of organic compounds are complex. Volatilization is the mass transfer of an organic compound from a specific medium (e.g., soil, water) into the air. The ability for this transfer or migration to occur will depend on other competing processes which may hinder this migration. For example, if a chemical is very soluble in water, it will be less likely to volatilize into the air. Environmental factors of importance include temperature, soil porosity, amount of water present in the soil, soil organic carbon content, and depth of contamination (Jury et al. 1983).

Generally, compounds with high vapor pressures or high Henry's law constants are expected to volatilize readily. Of the chemicals of concern at the SCP site, the chlorinated aliphatics (e.g., 1,1-dichloroethane, 1,2-dichloroethane, 1,1-dichloroethylene, 1,2-trans-dichloroethylene, methylene chloride, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene, and vinyl chloride) and some of the substituted simple aromatics (e.g., benzene, 1,2-dichlorobenzene, toluene, and total xylenes) have high vapor pressures and Henry's law constants greater than 1×10^{-3} atm-m^3/mol and, therefore, tend to volatilize readily from contaminated soils and water.

However, while the other chemicals of concern such as the pesticides, phthalate esters, PAHs, and PCBs have lower vapor pressures, volatilization can still be important (Mackay 1981).

Fugitive dust emissions could occur at the SCP site in areas that are unpaved or unvegetated. The potential for fugitive dust emission is primarily dependent on environmental factors such as particle size distribution, percent silt, moisture content, vegetative cover, and wind speed. The chlorinated aliphatics and substituted simple aromatics have a relatively low affinity for organic matter in the soil (as described by the K_{oc}) and moderate to high solubility in water. Though present at the site, these compounds are not expected to remain in suspended surface soils and, therefore, would not be present on dust transported from the site. The chlorinated pesticides, PAHs, PCBs, and phthalate esters, on the other hand, have relatively low water solubilities and higher K_{oc}s and would be expected to remain present in suspended soils.

The inorganic chemicals of concern can form insoluble precipitates with compounds found in soils or sorb onto the soil particles. These processes will result in the inorganic compounds remaining in suspended soil. As a result, these chemicals of concern could be transported in the air on fugitive dust.

3.1.2.4 Biodegradation and Biotransformation Processes

Biological and chemical processes that occur in the soil can be important in
determining the ultimate fate of the organic chemicals found at the SCP site.
These processes can produce more toxic and/or more mobile breakdown products.
In most cases, an organic chemical occurring in the natural environment is not
broken down immediately to carbon dioxide and water by a microorganism, but is
metabolized to an intermediate which is in turn further degraded. The
metabolites isolated depend primarily on the time at which the reaction is
stopped. In the course of the degradation of phenanthrene to low molecular
weight carboxylic acids by soil Pseudomonades (bacteria), a total of 24
different metabolites have been either isolated or proposed as intermediates
(Pucknat 1981). All of these intermediates are more water soluble than the
parent compound and are therefore more mobile. Many of the organic
intermediates are also more toxic. The extent and rates of these biological
and chemical reactions, however, are difficult to predict because of a limited
site-specific data base.

With respect to the chlorinated aliphatics, the state of knowledge in this
field has been reported in recent literature reviews (SAIC 1985, Smith and
Dragun 1984). Under anaerobic or oxygen depleted conditions, chlorinated
aliphatic chemicals (e.g., tetrachloroethylene, trichloroethylene) have been
found to undergo reductive dechlorination reactions, that is, reactions which
remove chlorine from and add hydrogen to the chemical (Bouwer et al. 1981,
Kobayashi and Rittman 1982). One exception to this is the aerobic degradation

of trichloroethylene in the presence of methane (Wilson and Wilson 1985). The transformation is sequential with, for example, tetrachloroethylene (an ethylene molecule with four chlorines) yielding trichloroethylene (an ethylene molecule with three chlorines), which in turn yields 1,1-dichloroethylene (an ethylene molecule with two chlorines) and ultimately vinyl chloride (an ethylene molecule with one chlorine) (Parsons et al. 1984, Cline and Viste 1984).

Chloroform has been found to degrade to methylene chloride in laboratory studies (Bouwer 1983). Wilson and coworkers (1981, 1983) did not observe this degradation in field studies and attributed this to the high mobility of chloroform in soils (i.e., it readily volatilizes or percolates to the ground water).

The nature and extent of degradation of chlorinated aliphatics, as well as the type and number of products, is highly dependent on soil conditions. Some investigators (Wilson et al. 1983, Schwarzenbach et al. 1983) found no transformation in soil, while others (Kleipfer et al. 1985, Parsons et al. 1984) found substantial transformation. It is possible that the vinyl chloride in the water table and till aquifers at the SCP site occurs as a result of the transformation of the unsaturated higher molecular weight chlorinated aliphatics (e.g., trichloroethylene). Vinyl chloride is stable with respect to further biological and/or chemical transformation and is likely to persist unless it has an opportunity to volatilize or leach from soil.

PCBs, which are comprised of mixtures of polychlorinated biphenyl congeners, may be metabolized by microorganisms present in the environment. Metabolism of one PCB congener will sequentially yield PCB congeners of lower molecular weight and greater solubility along with other metabolic byproducts such as PCB alcohols and/or ethers. Thus metabolism of PCBs in the environment will increase their mobility.

The PAHs present in soil can also be biodegraded. Factors which contribute to the degree to which biodegradation occurs include biodegradability rates, production of intermediates, and the effects of mixtures. In general, PAHs with 2 or 3 rings (i.e., phenanthrene) were more readily degraded than PAHs with 4 or more rings (i.e., pyrene) (McKenna and Heath 1976).

Based on the available site sampling data, it is difficult to determine if the chemicals that are present are byproducts of biotransformation (e.g., 1,1-dichloroethane from 1,1,1-trichloroethane) or if they are present due to past solvent refining and recovery activities. While some biodegradation may be occurring at this site, the high concentrations of many of the organic chemicals in soil could actually inhibit biotransformation from occurring by producing a toxic environment for microbial activity.

3.1.2.5 Bioaccumulation of Compounds Released From Surface Water and
          Sediments

The concentrations of organic and inorganic compounds in surface water and

sediments are governed, in part, by partitioning mechanisms which regulate the

amount of the compound which will be adsorbed by the sediments and the amount

remaining in the water column. This section discusses the relationship

between the presence of contaminants in the surface water and sediments and

the uptake of these contaminants by biota.

Sediments and the overlying water column may be viewed as a series of

compartments or layers. These layers may be viewed by their availability to

exchange, giving a deep or buried layer of sediment, and the active or

accessible layer of sediment on top interfacing with the water column (Reuber

et al. 1987). The properties regulating the initial partitioning of

contaminants among these compartments are based first on the properties of the

chemical. A chemical's solubility and corresponding hydrophobicity (lack of

affinity for water) are very important in determining the partitioning of the

compound. For organic compounds, such as PCBs, chlorinated pesticides, and

PAHs, the less soluble the compound and the higher its K_{ow} and K_{oc}, the more

likely it is to sorb to available organic carbon in the sediment or to be

taken up by biota such as fish (Karickhoff 1981).

The sediments provide an important role in reducing the amount of a chemical

available to the biota by acting as a 'sink' for the chemical. When chemical

concentrations in the water column are subsequently reduced, the chemical

sinks for organics and inorganics in the sediments then act as a source of the compound for aquatic exposure by slowly releasing the sorbed compound into the water column. Other processes that may also influence the transfer rates between sediment and water and/or biota are bioturbation, storm events and sedimentation. Bioturbation is defined as the mixing resulting from movement of the contaminated sediments by infauna or benthic foragers. This movement can either expose or bury the chemicals, and is dependent on the densities of interacting benthic organisms in addition to properties of the sediments.

Most of the hydrophobic organics (due to their high K_{oc}s) and inorganics that may enter Peach Island Creek from the site will quickly become adsorbed to organic and inorganic particulate matter, with a large proportion of the compounds deposited in the sediments. This contaminant burden usually remains relatively near the source, with concentrations decreasing with distance from the source. In fact, many of the chemicals of concern in sediments (e.g., trichloroethylene, toluene, Aroclor 1242) were detected at their highest levels immediately adjacent to the site. Leaching or biologic activity may then return a small portion of the chemicals adsorbed to sediments to the water column. By relative concentrations, inorganics and hydrophobic contaminants (e.g., PCBs, PAHs, chlorinated pesticides) in aquatic systems are generally highest in sediments, intermediate in biota and lowest in water (Neff 1985, Enk and Mathis 1977).

The bioconcentration factor (BCF) is the ratio of the concentration of a chemical in the organism to its concentration in the water (or other media).

MacKay (1982) correlated K_{ow} with BCF and determined a regression equation of $BCF=0.048\ K_{ow}$. Additionally, the more hydrophobic (higher K_{ow}) the compound, the slower it is released (depurated) from the organism. Several other factors such as steric properties of molecules and pharmacokinetic intercompartmental rate constants also affect uptake and depuration. Thus, K_{ow} should only be used to yield a relative estimate of the bioconcentration potential of a chemical.

Biotransformation reactions play an important role in determining actual body burdens of bioaccumulated chemicals. For example, Southworth et al. (1980) reported that many fish would be expected to bioconcentrate PAHs by factors of 10,000 based on K_{ow}. However, BCFs were about two orders of magnitude lower due to rapid metabolism of the parent compound.

Chemical transformation and degradation of the organics may occur through photo-oxidation, chemical oxidation and biological transformation. Most of these mechanisms require oxygen. Since oxygen is usually limited in deep sediments, a stable environment can result.

Uptake of metals in fish and invertebrates can occur both via the gills and from the diet (Biddinger and Gloss 1984). Chemical speciation is an important factor in the bioaccumulation of metals. For example, methylmercury is accumulated to a greater extent and eliminated more slowly in fish than inorganic mercury (Eisler 1987). Other factors that can affect the uptake of

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metals include salinity, hardness, and the presence of dissolved organic matter.

3.2 IDENTIFICATION OF EXPOSURE PATHWAYS

The purpose of this section is to identify the most important potential pathways through which humans may be exposed to the chemicals of concern in various media at the SCP site under current and possible future land use conditions of the site and surrounding area.

In a baseline risk assessment, exposure pathways are identified for the no-action alternative, that is, for the baseline condition assuming that no site remediation would occur or additional institutional controls be imposed.

There are three general routes through which individuals may be exposed to the chemicals of concern at the SCP site: inhalation, ingestion, and dermal absorption. The following sections describe the potential exposure pathways relevant to each environmental medium associated with the SCP site under present and future site use conditions. An identified pathway does not imply that exposures are actually occurring, only that the potential exists for the pathway to be complete. Potential exposure pathways are summarized in Table 3-1, with an indication of whether the pathway is complete. These exposure pathways will be discussed in the following sections.

TABLE 3-1

POTENTIAL PATHWAYS OF HUMAN EXPOSURE TO CONTAMINANTS AT THE SCP SITE UNDER CURRENT AND FUTURE SITE USE CONDITIONS

Exposure Medium	Potential Routes of Exposure	Potential Receptors	Pathway Complete
Air	Inhalation of volatile contaminants released from soil and/or fugitive dust	Current site use: Trespassers and nearby residents and workers on adjacent properties. Future site use: On-site workers	Yes. Soil contains wide variety of volatile compounds and potential also exists for wind erosion of exposed soil particles. No monitoring data are available, thus models need to be used. Although surface water also contains volatiles, this source is considered to be smaller and less important than the site itself.
Soil	Direct contact (dermal absorption, incidental ingestion)	Current site use: Trespassers Future site use: On-site workers	Current site use: Yes, although site is not readily accessible. Future site use: Yes.
Water Table and Till Aquifer Ground Water	Ingestion, inhalation of volatile organics, dermal absorption	Current site use: None Future site use: Possible on-site workers, workers in site area	Current site use: No, neither the water table nor till aquifer at the site, or in the area, is known to be used for domestic or industrial purposes. Future site use: All aquifers beneath the site are classified as potential drinking water sources. Use of these aquifers in the site vicinity is possible in the future. Potential exposures via inhalation and dermal absorption are not quantitatively estimated in this assessment.
Bedrock Aquifer Ground Water	Ingestion, inhalation of volatile organics, dermal absorption	Current site use: None onsite, residents and workers in site area Future site use: On-site workers	Current site use: No, for on-site, since bedrock aquifer at the site is not used for domestic or industrial purposes. Yes, for off-site, since bedrock aquifer is used as a public water supply. Future site use: Yes. Bedrock ground water could be used in the future, from either immediately under the site or in the vicinity of the site. Potential exposures via inhalation and dermal absorption are not quantitatively estimated in this assessment.
Surface Water/ Sediment	Dermal absorption, incidental ingestion Ingestion of aquatic biota	Current and future site use: Children, recreational users Current and future site use: Recreational fishing and crabbing	Current and future site use: Yes, however unlikely, due to inaccessibility of site within a generally commercial/industrial area, and site is not considered likely to provide major attraction for nearby residents or recreational users. Current and future site use: Highly unlikely, since neither fishing nor crabbing known to occur adjacent to site and recommendation against fish ingestion is in effect. Future site use: Possible, but creek adjacent to site is considered unlikely to provide major fishing/crabbing attraction.

3.2.1 <u>Air</u>

Since the surface material at the SCP site is flat, open, and sparsely

vegetated, exposed materials may be suspended into the air as a result of

mechanical forces (i.e., vehicles) or wind entrainment. As discussed in Dames

and Moore (1990), the surface fill at the site consists of soils mixed with a

wide variety of construction debris and earthen fill material. Although these

materials are of extremely variable composition and particle size, including

some concrete, wood, brick, crushed stone, sand, and gravel, exposed particles

may be suspended as a result of wind. For the purposes of this assessment,

the potential wind erosion of surface particles and their subsequent transport

to nearby residential areas was considered and was conservatively selected as

an air exposure pathway. There is currently no vehicular activity occurring

at the site and thus suspension of surface materials by mechanical forces is

not considered likely.

The soils at the SCP site and the water in Peach Island Creek can also act as

sources of volatile organic emissions to the air. Chemicals which volatilize

from soil or surface water can then be transported to surrounding areas.

Volatile emissions from on-site soil are expected to be of greater magnitude

than from Peach Island Creek (e.g., due to the greater magnitude of

contamination of soils and the relatively larger soil area source size) and

thus volatile emissions from soil only will be evaluated. Two possible air

pathways are therefore addressed under current site use conditions --

volatilization of chemicals from soil, and wind erosion and resuspension of exposed soil.

Under current site use conditions, nearby residents, site trespassers, and those who work daily in the area (e.g., at commercial areas such as those north of Peach Island Creek or at the racetrack) may be exposed to volatilized chemicals or suspended soils. In this assessment, exposures to both nearby residents and nearby workers are evaluated. Site trespassers are not evaluated because their potential inhalation exposures would be of much shorter duration and frequency than for nearby residents (short enough to compensate for higher on-site air levels). Individuals who may exercise at the racket club or visit the race track or businesses in the area would also be exposed considerably less frequently than residents or nearby off-site workers and, therefore, are not addressed.

Land in the site area is zoned for commercial or light industrial development, thus, future use might include development of the site as an industrial facility, or potentially, as a hotel or restaurant. It is unlikely that the site will be rezoned for residential development in the near future.[4] Therefore, under both current and possible future site use conditions, residential use of the site is not considered likely. Future on-site workers are, however, considered to be potential on-site receptors and are evaluated in this assessment. Potential inhalation exposures to off-site residents in

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[4]Personal communication with B. Nierstedt, Meadowlands Development Commission, June 29, 1988.

the future are assumed to be similar to those under current site and land use conditions. Therefore, under future land use conditions, potential inhalation exposures to possible future on-site workers are addressed in the quantitative risk assessment.

In summary, two general sources of air exposures are considered in this assessment, inhalation of volatilized organics and inhalation of suspended soils. Under current site use conditions, exposures to nearby residents and workers are considered and under future site use conditions exposures to on-site workers are considered.

3.2.2 Soil Contact

There are two main pathways through which an individual may be directly exposed to chemicals present in surface soil: incidental ingestion of soil and dermal absorption from contacted soil.

The SCP site is currently fenced on three sides, and the creek bounding the unfenced northeast side is over 20 feet wide. Although the site is not located near residential areas, facilities do exist near the site which are likely to be frequented by the public. In addition, it is plausible that some individuals may trespass on the site. In the future, if the site were developed, on-site workers could directly come into contact with chemicals present in surface soils. Furthermore, chemicals that may now be present in

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on-site surface and subsurface soils could be redistributed as a result of future site developments.

Thus for the soil exposure pathway, potential exposures to trespassers under current site use conditions, and to on-site workers under future site use conditions are considered. For potential future on-site workers, two different scenarios are evaluated. One scenario evaluates regular worker exposure to surface soils whereas the other evaluates potential infrequent exposures to subsurface soils (e.g., a construction or sewer line repair scenario).

3.2.3 Surface Water and Sediments

Potential pathways for exposure to surface water may be direct or indirect. Direct pathways involve incidental ingestion and dermal absorption of chemicals in surface water. Indirect pathways could include consumption of fish caught in Peach Island Creek.

Peach Island Creek and surrounding areas are located in waterfront recreational zones. Trappers of muskrat and fishermen in search of killifish (for use as bait), as well as older children from residences in the area may visit Peach Island Creek.[5] However, the area adjacent to the site is private industrial property and, therefore, not conducive to recreational use. The

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[5]E. Kinsevick, Laboratory Supervisor. HMDC. Personal Communication, June 29, 1988.

creek near the site is not likely to be used for boating because the creek is shallow and a culvert downstream of the site limits access to the creek adjacent to the site. Thus, human exposure to surface water in Peach Island Creek adjacent to the site is considered unlikely to occur. For similar reasons sediments in the creek adjacent to the site are not considered to be a likely source of human exposure and are not evaluated in the human health risk assessment. Since access to and use of Peach Island Creek could, however, change in the future, evaluation of this pathway should be reconsidered if more data become available in the future.

3.2.4 Ground Water

Fifty-seven wells have been identified within a two mile radius of the site; the reported well usages are for cooling and industrial water (51 wells), domestic water (2), lavatory water (1), and unknown (3). The wells of known depth are all installed in the bedrock aquifer, those of unknown depth are all industrial and/or cooling water wells. The three water wells of unknown usage are owned by Marathon Enterprises, Alpha Refining Co., and Stella Doro Co., Inc. The owners of the two domestic water wells are both also listed as customers of the Hackensack Water Company. Other wells may exist within the vicinity of the site, but have not been identified to date.

No wells have been identified in either the water table or till aquifers. However, Dames and Moore (1990) has reported that the water level in the till aquifer fluctuates in accord with a seven day cycle, with the highest levels

being recorded every Monday while the lowest levels are observed every Friday.

They have also indicated that this may be due to the influence of wells

operating Monday through Friday, and idling every weekend. This pattern,

therefore, indicates that waters are presently being withdrawn either directly

from the till aquifer or from an aquifer which is hydraulically connected to

the till aquifer which runs under the site. It also indicates that these

withdrawal(s) are sufficiently large enough and/or close enough to the site to

create this obvious weekly drawdown effect.

Both the water table and till aquifers are classified as GW2 by the State of

New Jersey, due in part to the hydrological connection to the bedrock aquifer,

which is a primary drinking water source. The GW2 classification refers to

groundwater intended for potable use without requiring treatment (e.g., for

VOCs) prior to use.

Based on this information, under current site conditions, potential exposures

to the water table, till, and bedrock aquifer ground water on the SCP site are

unlikely to occur and will not be evaluated. Potential exposures under

current site conditions to contaminants via use of the bedrock aquifer beyond

the SCP site is, however, likely since this aquifer is used as a public water

supply. Because off-site sampling data are unavailable for this aquifer, this

exposure pathway is not evaluated. Although the extent of contamination of

the bedrock aquifer in the vicinity of the site cannot be evaluated from the

currently available data, the presence of gross contamination in the overlying

water table and till aquifers and soils, along with the data from one well

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installed into the bedrock on site, indicate that migration of site contaminants to the bedrock aquifer is occurring and, in the absence of remediation, is likely to continue. Exposures to site-related contaminants by off-site users of the bedrock aquifer is possible, and may be occurring at present.

Under possible future site use conditions (e.g., development of the site for a hotel or restaurant), it is considered most likely that water would be purchased from the local water company rather than pumped from newly constructed ground water wells (as no wells, other than monitoring, currently exist on site). Although future use of on-site ground water for drinking water is considered unlikely, for the purposes of this assessment, a hypothetical and conservative future ground water use scenario is addressed. Properties which adjoin or are in the vicinity of the site are used for commercial and industrial purposes. It is also possible that a restaurant or hotel would be established on these properties in the future. To date, there are not sufficient data to estimate concentrations of chemicals in off-site areas based on on-site concentrations. Therefore, the scenario evaluated in this assessment assumes the site will be developed for a restaurant or hotel, and that an individual employed on-site will regularly ingest on-site ground water for the duration of his or her employment. Each of the three aquifers (water table, till, and bedrock) will be evaluated separately for this pathway.

It should be noted that the bedrock aquifer may be considered the most likely future source of ground water at the site for the reasons identified above. Furthermore, the potential risks associated with future on-site worker ingestion of bedrock aquifer ground water may be the most comparable to potential risks associated with current and future off-site ingestion of ground water in nearby locations, based on groundwater use information currently available. Although chemical concentrations in the bedrock aquifer may be slightly lower off-site, it should be noted that off-site residents consuming water from this aquifer are likely to ingest a greater quantity of ground water per day (on a greater number of days per year, over a longer period of time) than the worker ingestion scenario that is evaluated.

Depending on which of the above two considerations has the greater impact, the risks associated with potential current or future off-site exposures may be greater or smaller than the risks associated with the future on-site worker exposure scenario evaluated in this assessment.

3.2.5 Consumption of Fish

Some limited fishing may occur in Peach Island Creek. Although the Hackensack Meadowlands Development Commission (HMDC) has publicly recommended that fish from the lower Hackensack River not be consumed,[6] and although the state has

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[6]E. Konsevick, Laboratory Supervisor. HMDC. Personal communication, June 29, 1988.

an advisory against consuming eels from the Hackensack River,[7] it is likely

that some recreational fishing is occurring in this area despite these

recommendations. The HMDC anticipates that the water quality in Berry's Creek

(approximately one-half mile downstream from the SCP site to the west) will

improve because the sewage treatment plant that discharged into the creek has

been closed. The HMDC also anticipates that more fish will move up Berry's

Creek in the future and that recreational use of Berry's Creek and possibly

Peach Island Creek may also increase. Such use may include fishing and

crabbing. Because only limited data regarding the impact of the site on biota

are available at this time, exposures via fish or crab consumption under

future site use conditions are not evaluated in this assessment.

3.3 EXPOSURE POINT CONCENTRATIONS

In this section, concentrations of the selected chemicals of concern

to which an individual may be exposed are presented for the exposure pathways

noted above. In estimating exposure point concentrations (and exposures), two

cases are considered in this assessment: an average case and, in accordance

with recent USEPA guidance (USEPA 1989a), a reasonable maximum exposure (RME)

case. The average case is intended to represent the exposure of a typical

individual. The RME scenario, which is specifically recommended and described

in USEPA (1989a), is intended to reflect a conservative exposure case. For

the purposes of this risk assessment, a different set of exposure point

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[7]New Jersey Fish and Game Department. Personal communication, December
27, 1989.

concentrations are used for each exposure case. In general, geometric mean concentrations are used for the average case while maximum detected concentrations are used for the RME case. It should be noted that USEPA (1989a) directs that exposure point concentrations for the RME case be based on the upper 95 percent confidence limit on the arithmetic average. Since USEPA's risk assessment guidelines for Superfund (USEPA 1989a) were published after this project was underway, and after exposure point concentrations had been calculated, the upper 95 percent confidence limits on the arithmetic averages were not calculated for the RME case. For some data sets with a large degree of variability, however, the upper 95 percent confidence limit on the mean can exceed the maximum, in which case USEPA (1989a) directs that the maximum detected value be used for the RME case. The net effect in the majority of cases of using maximum detected concentrations rather than upper 95 percent confidence limits on the means is higher estimates of exposure and ultimately risk. For the purposes of assessing a conservative, upper bound exposure case, use of the maximum concentrations is considered reasonable.

3.3.1 Contact With On-Site Surface Soil

Under this exposure scenario, the concentrations of chemicals of concern in surface soil presented in Table 2-2 are assumed to be representative of concentrations of chemicals to which trespassers and potential future on-site workers may be exposed. For the future construction/sewer repair scenario, the soil concentrations measured in the saturated fill (5-6 feet deep) will be used. Two sets of exposure point concentrations will be used to evaluate

potential risks: the geometric mean concentration will be used for the average exposure case and the maximum concentrations detected will be used for the RME case.

For the two classes of PAHs, carcinogenic and noncarcinogenic, exposure point concentrations were determined by summing the results for each member of the class to obtain total carcinogenic and total noncarcinogenic PAH concentrations. The classification for each PAH (carcinogenic or noncarcinogenic) was based on the IARC PAH classification scheme (see Table 2-3 in Section 2.3.1.1). Similarly, the results for the Arochlors were summed to provide estimates of total PCB concentrations in soil. These approaches are in accordance with current standard USEPA Superfund site risk assessment procedures.

3.3.2   <u>Inhalation of Airborne Chemicals by Nearby Residents and On-Site Workers</u>

In the absence of air monitoring data, to evaluate the potential exposures to nearby residents and on-site workers through inhalation, air modeling was performed. A time-dependent mathematical model (Hwang 1986) summarized in Appendix B was used to calculate the emission rates of organic contaminants due to volatilization from soils at the SCP site. To reflect average chemical concentrations in the potential soil source area from which emissions could occur, the soil concentrations used in the Hwang (1986) model were geometric means of all the soil sampling results (i.e., from all depths sampled). A fugitive dust emissions model (Cowherd et al. 1984) which is also summarized

in Appendix B was used to calculate the emission rate of contaminants due to wind erosion.

Once emission rates of volatilized chemicals and suspended soils were estimated, air models were used to predict potential concentrations at the receptor points. Concentrations in off-site areas were predicted using the USEPA-approved Industrial Source Complex Long Term (ISCLT) dispersion model which is summarized in Appendix B. Concentrations on-site (i.e., for potential future on-site workers) were predicted using a box model, also summarized in Appendix B.

Tables 3-2 and 3-3 summarize the estimated chemical emission rates and estimated on- and off-site air concentrations due to volatilization and wind erosion. For air concentrations due to dust suspension it was assumed that concentrations will remain constant for the period of exposure. For chemicals that may volatilize, air concentrations were derived for the time period during which emissions would occur (i.e., up until the chemical was essentially depleted from soil). For the VOCs, this time period was often less than five years, in which case the air concentrations were averaged over five or less years (rather than for example a 70-year period during 65 years of which emissions would be negligible).

TABLE 3-2

ESTIMATED AIR CONCENTRATIONS OF CHEMICALS
DUE TO VOLATILIZATION FROM SOIL AT THE SCP SITE

Chemical	Geometric Mean Concentration in Soil (ug/kg) (a)	Estimated Emission Rate (g/cm2-sec) (b)	Air Concentration (mg/m3)		
			On-Site (c)	Off-Site Maximum in Industrial Area (d)	Off-Site Maximum in Residential Area (d)
Aldrin	4.99	1.61E-16	1.29E-08	9.54E-10	4.53E-11
Benzene	120.0	7.32E-13	5.85E-05	4.34E-06	2.06E-07
Bis (2-ethylhexyl)phthalate	6,371.0	1.60E-13	1.27E-05	9.45E-07	4.49E-08
Butyl benzyl phthalate	871.3	3.42E-14	2.73E-06	2.02E-07	9.62E-09
Carcinogenic PAHs	7,073.1	1.64E-14	1.31E-06	9.70E-08	4.61E-09
Noncarcinogenic PAHs	3,633.7	2.01E-12	1.60E-04	1.19E-05	5.65E-07
Chlorobenzene	181.1	3.58E-13	2.86E-05	2.12E-06	1.01E-07
Chloroform	80.1	4.30E-12	3.43E-04	2.55E-05	1.21E-06
1,2-Dichlorobenzene	275.7	1.54E-13	1.23E-05	9.11E-07	4.33E-08
1,1-Dichloroethane	112.0	6.53E-12	5.22E-04	3.87E-05	1.84E-06
1,2-Dichloroethane	118.0	5.13E-13	4.10E-05	3.04E-06	1.44E-07
1,1-Dichloroethylene	9.6	3.90E-12	3.11E-04	2.31E-05	1.10E-06
1,2-trans-Dichloroethylene	69.1	2.98E-12	2.38E-04	1.77E-05	8.39E-07
Dieldrin	11.6	4.83E-16	3.86E-08	2.86E-09	1.36E-10
Di-n-butylphthalate	1,033.0	3.31E-15	2.64E-07	1.96E-08	9.32E-10
Ethyl benzene	440.7	3.88E-13	3.10E-05	2.30E-06	1.09E-07
Methylene chloride	315.4	2.40E-11	1.91E-03	1.42E-04	6.74E-06
Methyl ethyl ketone	561.5	9.38E-13	7.49E-05	5.56E-06	2.64E-07
Xylenes (e)	2,597.0	5.23E-12	4.18E-04	3.10E-05	1.47E-06
PCBs	2,253.0	2.53E-13	2.02E-05	1.50E-06	7.12E-08
Phenol	149.2	2.28E-13	1.82E-05	1.35E-06	6.43E-08
Tetrachloroethylene	845.9	2.20E-12	1.76E-04	1.31E-05	6.21E-07
Toluene	1,322.0	5.86E-12	4.68E-04	3.47E-05	1.65E-06
1,1,2,2-Tetrachloroethane	24.0	1.34E-14	1.07E-06	7.94E-08	3.77E-09
1,1,1-Trichloroethane	136.0	2.37E-12	1.89E-04	1.40E-05	6.67E-07
Trichloroethylene	488.0	2.28E-11	1.82E-03	1.35E-04	6.41E-06
Vinyl Chloride	6.54	1.28E-11	1.02E-03	7.58E-05	3.60E-06

(a) Based on soil data obtained from all depth ranges sampled (unsaturated fill, saturated fill, top of clay, within the clay). The chemicals shown in this table are those detected in site soil and which are expected to be able to volatilize into air (i.e., inorganic chemicals were not included).

(b) Emission estimates based on the Hwang (USEPA 1986) soil volatilization model.

(c) On-site air concentrations based on the Box model (see Appendix B).

(d) Off-site air concentrations based on the ISCLT model (see Appendix B). The maximum annual average air concentrations predicted for a unit emission rate of 1 g/m2-sec for an industrial area (i.e., a nearby worker) and a residential area (i.e., a nearby resident) were 592,352 ug/m3 and 28,153 ug/m3, respectively. The maxima were located 200 m and 1,000 m to the NE of the site center, respectively.

(e) Chemical-specific parameters based on m-xylene.

TABLE 3-3

ESTIMATED AIR CONCENTRATIONS OF CHEMICALS
DUE TO WIND EROSION FROM SURFACE SOILS AT THE SCP SITE

Chemical	Geometric Mean Mass Fraction in Surface Soils (g/g) (a)	Estimated Emission Rate (g/m2-sec) (b)	Air Concentration (mg/m3)		
			On-Site (c)	Off-Site Maximum in Industrial Area (d)	Off-Site Maximum in Residential Area (d)
Semi-Volatile Compounds					
Bis (2-ethylhexyl)phthalate	3.36E-05	6.15E-10	4.91E-06	3.64E-07	1.73E-08
Butyl benzyl phthalate	1.54E-06	2.83E-11	2.26E-07	1.67E-08	7.95E-10
Di-n-butyl phthalate	3.08E-06	5.64E-11	4.50E-07	3.34E-08	1.59E-09
Di-n-octyl phthalate	1.57E-06	2.87E-11	2.29E-07	1.70E-08	8.08E-10
Carcinogenic PAHs	8.47E-06	1.55E-10	1.24E-06	9.18E-08	4.36E-09
Noncarcinogenic PAHs	8.97E-06	1.64E-10	1.31E-06	9.72E-08	4.62E-09
Phenol	1.45E-07	2.65E-12	2.12E-08	1.57E-09	7.47E-11
2-Chloronaphthalene	1.74E-07	3.18E-12	2.54E-08	1.89E-09	8.96E-11
1,2-Dichlorobenzene	5.43E-07	9.94E-12	7.94E-08	5.89E-09	2.80E-10
2,4-Dimethylphenol	1.88E-07	3.44E-12	2.75E-08	2.04E-09	9.69E-11
Pesticides/PCBs					
PCBs	3.95E-06	7.23E-11	5.77E-07	4.28E-08	2.04E-09
Dieldrin	1.70E-07	3.11E-12	2.48E-08	1.84E-09	8.76E-11
Aldrin	4.40E-08	8.05E-13	6.43E-09	4.77E-10	2.27E-11
Inorganic Chemicals					
Antimony	3.80E-06	6.95E-11	5.55E-07	4.12E-08	1.96E-09
Arsenic	8.12E-06	1.49E-10	1.19E-06	8.80E-08	4.18E-09
Cadmium	6.10E-06	1.12E-10	8.92E-07	6.61E-08	3.14E-09
Chromium	7.85E-05	1.44E-09	1.15E-05	8.51E-07	4.04E-08
Cyanide	1.85E-06	3.39E-11	2.70E-07	2.01E-08	9.53E-10
Lead	4.90E-04	8.97E-09	7.16E-05	5.31E-06	2.52E-07
Mercury	1.40E-06	2.56E-11	2.05E-07	1.52E-08	7.21E-10
Nickel	1.22E-05	2.23E-10	1.78E-06	1.32E-07	6.29E-09
Selenium	4.90E-07	8.97E-12	7.16E-08	5.31E-09	2.52E-10
Silver	1.10E-06	2.01E-11	1.61E-07	1.19E-08	5.67E-10
Zinc	3.98E-06	7.28E-11	5.82E-07	4.31E-08	2.05E-09

(a) Based on soil data obtained from shallow soil depths. The chemicals shown in this table were those detected in shallow soil at the site and which are expected to persist on suspended soil particles (i.e., volatile organic chemicals were not included).

(b) Emission estimates based on Cowherd et al. (1984) wind erosion model.

(c) On-site sir concentrations based on the Box model (see Appendix B).

(d) Off-site air concentrations based on the ISCLT model (see Appendix B). The maximum annual average air concentrations predicted for a unit emission rate of 1 g/m2-sec for an industrial area (i.e., a nearby worker) and a residential area (i.e., a nearby resident) were 592,352 ug/m3 and 28,153 ug/m3, respectively. The maxima were located 200 m and 1000 m to the NE of the site center, respectively.

3.3.3 Ingestion of Ground Water

The concentrations of the chemicals of concern in ground water presented in
Tables 2-8, 2-9, and 2-10 (water table, till, and bedrock aquifers,
respectively) are considered to be representative of potential future exposure
point concentrations. The geometric mean concentrations were used for the
average case while the maximum detected concentrations were used for the RME
case.

3.4 SUMMARY OF POTENTIAL EXPOSURE PATHWAYS TO BE QUANTITATIVELY EVALUATED

The potential exposure pathways to be evaluated which are considered most
likely to be of concern to human health under current site use conditions are:
1) contact with on-site surface soils by trespassers, 2) inhalation of
volatilized organics by nearby (off-site) residents and workers, and 3)
inhalation of suspended soil by nearby (off-site) residents and workers.

Under future site use conditions, the pathways selected for detailed
evaluation are: 1) contact with on-site surface soils by future site workers,
2) contact with on-site subsurface soils by future site construction workers,
3) inhalation of volatilized organics by future on-site workers, 4) inhalation
of suspended soil by future on-site workers, and 5) ingestion of ground water
by future on-site workers. These exposures will be assessed quantitatively in
Section 5.0. (Exposures via inhalation and dermal absorption of chemicals in
ground water are not quantitatively evaluated.)

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It should be noted that some of these pathways may occur simultaneously to a single receptor. For example, under current site use conditions, a nearby resident may not only inhale volatilized organics released from site soil, but also inhale suspended site soil. Similarly, under future site use conditions, an on-site worker could potentially be exposed through four pathways: contact with on-site surface soil; inhalation of volatilized organics; inhalation of suspended soil; and ingestion of ground water. Thus in this assessment, risks estimated for individual pathways will be presented as well as risks for a potential receptor that may be exposed via more than one pathway.

Other potential pathways of exposure which may exist but which will not be evaluated in the assessment (e.g., due to insufficient sampling data) include: incidental ingestion of surface water and sediments from Peach Island Creek, ingestion of fish or shellfish caught from Peach Island Creek, and ingestion of off-site ground water.

4.0 HAZARD IDENTIFICATION

In this section, brief summaries of the potential health effects of each selected chemical of concern are presented. In addition, the health effects criteria (i.e., dose-response values) that will be used to evaluate human health risks are provided.

4.1 CLASSIFICATION OF HEALTH EFFECTS

For risk assessment purposes, individual pollutants are separated into two categories of chemical toxicity depending on whether they exhibit the potential for noncarcinogenic or carcinogenic effects in humans.

4.1.1 Health Effects Criteria For Carcinogenic Effects

For chemicals that exhibit carcinogenic effects, USEPA as well as other scientific authorities recognize that one or more molecular events can evoke changes in a single cell or a small number of cells that can lead to tumor formation. This is the non-threshold theory of carcinogenesis which purports that any level of exposure to a carcinogen can result in some finite possibility of generating the disease. Generally, regulatory agencies assume the non-threshold hypothesis for carcinogens in the absence of information concerning the mechanisms of carcinogenic action for the chemical.

USEPA's Carcinogen Assessment Group (CAG) has developed slope factors (i.e., cancer potency factors or dose-response values) for estimating excess lifetime cancer risks associated with various levels of lifetime exposure to potential human carcinogens. The slope factor [in units of (mg/kg body weight/day)$^{-1}$] is a number which, when multiplied by the lifetime average daily dose of a potential carcinogen (in mg/kg body weight/day), yields the upper bound lifetime excess cancer risk associated with exposure at that dose. Upper bound is a term used by USEPA to reflect the conservative nature of the slope factors; risks estimated using slope factors are considered unlikely to underestimate actual risks but they may overestimate actual risks for a given exposure. This multiplication approach can be used for low doses corresponding to cancer risks lower than 10^{-2} (one in one hundred). Excess lifetime cancer risks are generally expressed in scientific notation and are probabilities. An excess lifetime cancer risk of 1×10^{-6} (one in one million), for example, represents the incremental probability that an individual will develop cancer as a result of exposure to a carcinogenic chemical over a 70-year lifetime under specified exposure conditions. USEPA has suggested developing remedial alternatives for cleanup of Superfund sites to achieve total excess lifetime cancer risks ranging from no more than 10^{-4} (one in ten thousand) to 10^{-6} (one in one million) (USEPA 1990).

In practice, slope factors are derived from the results of human epidemiology studies or chronic animal bioassays. For the latter, data from animal studies are fitted to the linearized multistage model and a dose-response curve is obtained. The 95th percentile upper confidence limit slope of the dose-

response curve is subjected to various adjustments, and an interspecies scaling factor is applied to conservatively derive the slope factor for humans. Thus, the actual risks associated with exposure to a potential carcinogen quantitatively evaluated based on animal data are not likely to exceed the risks estimated using these slope factors, but they may be much lower. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an ad-hoc basis. These models provide rough, but plausible, estimates of the upper limits on lifetime risk.

In addition, there are varying degrees of confidence in the weight of evidence for carcinogenicity of a given chemical. USEPA (1986b) has proposed a system for characterizing the overall weight of evidence for a chemical's carcinogenicity based on the availability of animal, human, and other supportive data. The weight-of-evidence classification is an attempt to determine the likelihood that an agent is a human carcinogen and thus qualitatively affects the estimation of potential health risks. Three major factors are considered in characterizing the overall weight of evidence for carcinogenicity: (1) the quality of evidence from human studies and (2) the quality of evidence from animal studies which are combined into a characterization of the overall weight of evidence for human carcinogenicity, and then (3) other supportive information which is assessed to determine whether the overall weight of evidence should be modified. USEPA's final classification of the overall weight of evidence has the following five categories:

Group A--Human Carcinogen

This category indicates that there is sufficient evidence from human epidemiological studies to support a causal association between an agent and cancer.

Group B--Probable Human Carcinogen

This category generally indicates that there is at least limited evidence from epidemiological studies of carcinogenicity to humans (Group B1) or that, in the absence of adequate data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).

Group C--Possible Human Carcinogen

This category indicates that there is limited evidence of carcinogenicity in animals in the absence of data on humans.

Group D--Not Classified

This category indicates that the evidence for carcinogenicity in animals is inadequate.

Group E--No Evidence of Carcinogenicity to Humans

This category indicates that there is no evidence for carcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies.

Slope factors are developed based on epidemiological or animal bioassay data for a specific route of exposure, either oral or inhalation. For some chemicals, such as tetrachloroethylene and chloroform, sufficient data are available to develop route-specific slope factors for inhalation and ingestion. In accordance with recent USEPA (1989a) guidance, slope factors were only used for the route of exposure they were based on (e.g., oral slope factors were not used to evaluate the inhalation route of exposure). The only exception to this rule was that oral slope factors were used to evaluate

dermal exposure, as directed in USEPA's recent Superfund risk assessment guidance (USEPA 1989a).

4.1.2 Health Effects Criteria For Noncarcinogenic Effects

For chemicals that exhibit noncarcinogenic (e.g., systemic) effects, many authorities consider organisms to have repair and detoxification capabilities that must be exceeded by some critical concentration (threshold) before the health effect is manifested. For example, an organ can have a large number of cells performing the same or similar functions that must be significantly depleted before the effect on the organ is seen. This threshold view holds that a range of exposures from just above zero to some finite value can be tolerated by the organism without an appreciable risk of adverse effects.

Health criteria for chemicals exhibiting noncarcinogenic effects for use in risk assessment are generally developed using USEPA reference doses (RfDs) developed by the RfD Work Group. In accordance with recent USEPA (1989a) guidance, only USEPA-approved RfDs are used in this assessment. The RfD values (and slope factors) were obtained from the USEPA Integrated Risk Information System (IRIS) or USEPA Health Effects Assessment Summary Tables (HEASTs) where available. In some cases, such as for PCBs, oral RfDs were obtained from other sources or were derived and approved for use by USEPA's Environmental Criteria and Assessment Office. The RfD is expressed in units of mg chemical/kg body weight/day. In general, the RfD is an estimate of an average daily exposure to an individual (including sensitive individuals)

below which there will not be an appreciable risk of adverse health effects. The RfD is derived using conservative safety factors (e.g., to adjust from animals to humans and to protect sensitive subpopulations) to ensure that it is unlikely to underestimate the potential for adverse noncarcinogenic effects to occur. The purpose of the RfD is to provide a benchmark against which other exposures (e.g., those projected from human exposure to various environmental conditions) might be compared. Exposures that are significantly higher than the RfD may indicate that an inadequate margin of safety could exist for exposure to that substance and that an adverse health effect could occur.

4.2 RANGE OF POTENTIAL HEALTH EFFECTS FOR SELECTED CHEMICALS OF CONCERN

This section of the BRA presents a brief summary of the critical human health effects associated with long-term (chronic) exposure to each of the selected chemicals of concern. Although exposures to chemicals at Superfund sites are not generally associated with adverse effects from high level short term exposures (acute effects), this section includes information on acute effects for completeness. In addition, the available health effects criteria for use in risk assessment (slope factors and RfDs) for each chemical of concern are presented. The data that provide the basis for the health criteria values are also discussed. Information on potential human health effects is primarily obtained from information in USEPA reports (e.g., Health Effects Assessment Documents, Health Effects Criteria Documents, Health Assessment Documents, Health and Environmental Effects Profiles, and Ambient Water Quality Criteria

Documents), and from published and unpublished toxicological and epidemiological studies.

Some chemicals exhibit different health effects depending upon the route of exposure (e.g., inhalation versus oral). For example, a chemical may be associated with an increased risk of cancer when inhaled but may have no evidence of carcinogenicity when ingested [e.g., nickel and cadmium (USEPA 1987a,b)]. For these chemicals, route-specific health effects criteria where available are used, such as oral- and inhalation-specific slope factors and RfDs. Table 4-1 and 4-2 present the health effects criteria for oral and inhalation exposure, respectively, which were used to assess risks in this BRA.

4.2.1 Organic Chemicals

Aldrin

Aldrin is absorbed following inhalation exposure; between 20-50% of the inhaled vapor is absorbed and retained (Beyermann and Eckrich 1973, Shell 1984). Absorption also occurs following ingestion (Farb et al. 1973, Heath and Vandekar 1964, Hunter and Robinson 1967, 1969, Iatropoulos et al. 1975) and dermal exposure (Feldmann and Maibach 1974, Sundaram et al. 1978a,b). It is metabolically converted to dieldrin in fatty tissues (ACGIH 1986) and these two insecticides are considered to have similar chemical and toxic effects (USEPA 1988). Acute symptoms of aldrin intoxication in humans and animals following ingestion or inhalation indicate CNS stimulation manifested

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TABLE 4-1

ORAL CRITICAL TOXICITY VALUES FOR SELECTED CHEMICALS OF CONCERN

Chemical	Chronic RfD (mg/kg/day) [Uncertainty Factor] (a)	Source	Subchronic RfD (HEAST) (mg/kg/day) [Uncertainty Factor] (a)	Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Category (b)	Source
Aldrin	3E-05 [1,000]	IRIS	3E-05 [1,000]	1.7E+01	B2	IRIS
Antimony	4E-04 [1,000]	IRIS	4E-04 [1,000]	--	--	--
Arsenic	1E-03 [1]	HEAST (c)	1E-03 [1]	2.0E+00	A	(d)
Benzene	--*	--	--	2.9E-02	A	IRIS
Bis(2-ethylhexyl)phthalate	2E-02 [1,000]	IRIS	2E-02 [1,000]	1.4E-02	B2	IRIS
Butyl benzyl phthalate	2E-01 [1,000]	IRIS	2E+00 [100]	--	C	IRIS
Cadmium	5E-04 [10] (water)	IRIS	--	--	--	--
	1E-03 [10] (e)	IRIS	--			
Carcinogenic PAHs (as Benzo[a]pyrene)	--	--	--	1.15E+01	B2	(f)
Chlorobenzene	2E-02 [1,000]	IRIS	2E-01 [100]	--*	--	--
Chloroform	1E-02 [1,000]	IRIS	1E-02 [1,000]	6.1E-03	B2	IRIS
2-Chloronaphthalene	--	--	--	--	--	--
Chromium (III)	1E+00 [1,000]	IRIS	--.	--	--	--
Chromium (VI)	5E-03 [500]	IRIS	--	--	--	--
Cyanide	2E-02 [500]	IRIS	2E-02 [500]	--	--	--
1,2-Dichlorobenzene	9E-02 [1,000]	IRIS	9E-01 [100]	--	--	--
1,1-Dichloroethane	1E-01 [1,000]	HEAST	1E+00 [100]	9.1E-02	B2	HEAST
1,2-Dichloroethane	--	--	--	9.1E-02	B2	IRIS
1,1-Dichloroethylene	9E-03 [1,000]	IRIS	9E-03 [1,000]	6E-01	C	IRIS
trans-1,2-Dichloroethylene	2E-02 [1,000]	IRIS	--	--	--	--
Dieldrin	5E-05 [100]	IRIS	5E-05 [100]	1.6E+01	B2	IRIS
2,4-Dimethylphenol	--*	--	--	--	--	--
Di-n-butyl phthalate	1E-01 [1,000]	IRIS	1E+00 [100]	--	--	--
Di-n-octyl phthalate	--	--	--	--	--	--
Ethylbenzene	1E-01 [1,000]	IRIS	1E+00 [100]	--	--	--
Isophorone	2E-01 [1,000]	IRIS	2E+00 [100]	4.1E-03*	C	HEAST
Lead	--	--	--	--	B2	IRIS
Mercury (inorganic)	3E-04 [1,000]	HEAST	3E-04 [1,000]	--	--	--
Methylene chloride	6E-02 [100]	IRIS	6E-02 [100]	7.5E-03	B2	IRIS
Methyl ethyl ketone	5E-02 [1,000]	IRIS	5E-01 [100]	--	--	--
Nickel	2E-02 [300]	IRIS	2E-02 [300]	--	--	--
Nitrobenzene	5E-04 [10,000]	IRIS	5E-03 [1,000]	--*	--	--
Noncarcinogenic PAHs (as Naphthalene)	4E-01 [100]	HEAST	4E-01 [100]	--	--	--
Phenol	6E-01 [100]	IRIS	6E-01 [100]	--	--	--
PCBs	1E-04 [100]	(g)	--	7.7E+00	B2	IRIS
Selenium	3E-03 [15]	HEAST	4E-03 [100]	--	--	--
Silver	3E-03 [2]	IRIS	--	--	--	--
1,1,2,2-Tetrachloroethane	4.6E-04	HA 1988 (h)	--	2E-01	C	IRIS
Tetrachloroethylene	1E-02 [1,000]	IRIS	1E-01 [100]	5.1E-02*	B2	HEAST
Toluene	3E-01 [100]	IRIS	4E-01 [100]	--	--	--
1,1,1-Trichloroethane	9E-02 [1,000]	IRIS	9E-01 [100]	--	--	--
Trichloroethylene	7.35E-03 [1,000]	HA 1985 (h)	--	1.1E-02	B2	HEAST
Vinyl chloride	--	--	--	2.3E+00	A	HEAST
Xylenes (o,m &p)	2E+00 [100]	IRIS	4E+00 [100]	--	--	--
Zinc	2E-01 [10]	HEAST	2E-01 [10]	--	--	--

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
. A 10-fold factor to account for the variation in sensitivity among the members of the human population;
. A 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
. A 10-fold factor to account for uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and,
. A 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.
Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.
(b) Weight of evidence classification scheme for carcinogens: A--Human Carcinogen, sufficient evidence from human epidemiological studies; B1--Probable Human Carcinogen, limited evidence from epidemiological studies and adequate evidence from animal studies; B2--Probable Human Carcinoagen, inadequate evidence from epidemiological studies and adequate evidence from animal studies; C-- Possible Human Carcinogen, limited evidence in animals in the absence of human data; D--Not Classified as to human carcinogenicity; and E--Evidence of Noncarcinogenicity.
(c) The oral RfD is being reconsidered by the RfD workgroup.
(d) EPA 1988. Special report on Ingested Inorganic Arsenic. Skin Cancer; Nutritional Essentiality. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, D.C. EPA/625/3-87/013F. July 1988. Approved by Dr. Hurst at EPA ECAO.
(e) In accordance with EPA guidance, the listed cadmium RfD is used for exposures to food and other nonaqueous materials (i.e., soil).

TABLE 4-1

ORAL CRITICAL TOXICITY VALUES FOR SELECTED CHEMICALS OF CONCERN

(f) Health Effects Assessment for Benzo(a)pyrene. Environmental Criteria and Assessment Office. Cincinnati, Ohio. September 1984. EPA 540/1-86/022. Approved by Dr. Hurst at EPA ECAO.

(g) Calculated by Clement Associates based on data in Barsotti, D.A., and Van Miller, J.P. 1984. Accumulation of commercial polychlorinated biphenyl mixture (Aroclor 1016) in adult rhesus monkeys and their nursing infants. Pathology, 30(1984) 31-44. Received site specific approval from Dr. Pei-Fung Hurst at EPA Environmental Criteria and Assessment Office on January 30, 1990.

(h) Provided by Dr. Pei-Fung Hurst at EPA Environmental Criteria and Assessment Office on January 30, 1990.

    * = Review Pending.
  -- = Criterion has not been developed for this chemical.
 IRIS = Chemical files of the Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.
  HA = Health Advisory.

TABLE 4-2

INHALATION CRITICAL TOXICITY VALUES FOR SELECTED CHEMICALS OF CONCERN

Chemical	RfD (mg/kg/day) [Uncertainty Factor] (a)	Source	Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Category (b)	Source
Aldrin	--	--	1.7E+01	B2	IRIS
Antimony	--	--	--.	--	--
Arsenic	--	--	5.0E+01	A	IRIS
Benzene	--*	--	2.9E-02	A	IRIS
Bis(2-ethylhexyl)phthalate	--	--	--	B2	IRIS
Butyl benzyl phthalate	--	--	--	C	IRIS
Cadmium	--*	--	6.1E+00	B1	IRIS
Carcinogenic PAHs (as Benzo[a]pyrene)	--	--	--	B2	IRIS
Chlorobenzene	5E-03 [10,000]	HEAST	--*	--	--
Chloroform	--*	--	8.1E-02	B2	IRIS
2-Chloronaphthalene	--	--	--	--	--
Chromium (III)	--*	--	--	--	--
Chromium (VI)	--*	--	4.1E+01	A	IRIS
Cyanide	--	--	--	--	--
1,2-Dichlorobenzene	4E-02 [1,000]	HEAST	--	--	--
1,1-Dichloroethane	1E-01 [1,000]	HEAST	--	--	--
1,2-Dichloroethane	--	--	9.1E-02	B2	IRIS
1,1-Dichloroethylene	--*	--	1.2E+00	C	IRIS
trans-1,2-Dichloroethylene	--	--	--	--	--
Dieldrin	--	--	1.6E+01	B2	IRIS
2,4-Dimethylphenol	--	--	--	--	--
Di-n-butyl phthalate	--	--	--	--	--
Di-n-octyl phthalate	--	--	--	--	--
Ethylbenzene	--	--	--	--	--
Isophorone	--	--	--*	C	HEAST
Lead	--	--	--	B2	IRIS
Mercury (inorganic)	--*	--	--	--	--
Methylene chloride	8.6E-01 [100] (c)	HEAST	1.4E-02	B2	IRIS
Methyl ethyl ketone	9E-02 [1,000]	HEAST	--	--	--
Nickel	--	--	1.7E+00 (d)	A	IRIS
Nitrobenzene	6E-04 [10,000]	HEAST	--*	--	--
Noncarcinogenic PAHs (as Naphthalene)	--	--	--	--	--
Phenol	--*	--	--	--	--
PCBs	--	--	--	B2	IRIS
Selenium	1E-03 [10]	HEAST	--	--	--
Silver	--	--	--	--	--
Toluene	5.7E-01 [100] (c)	HEAST	--	--	--
1,1,2,2-Tetrachloroethane	--	--	2E-01	C	IRIS
Tetrachloroethylene	--	--	3.3E-03*	B2	HEAST
1,1,1-Trichloroethane	3E-01 [1,000]	HEAST	--	--	--
Trichloroethylene	--	--	1.7E-02	B2	HEAST
Vinyl chloride	--	--	2.95E-01	A	HEAST
Xylenes (o,m &p)	8.6E-02 [100] (c)	HEAST	--	--	--
Zinc	--	--	--	--	--

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
   . A 10-fold factor to account for the variation in sensitivity among the members of the human population;
   . A 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
   . A 10-fold factor to account for uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and,
   . A 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.
   Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.
(b) Weight of evidence classification scheme for carcinogens: A--Human Carcinogen, sufficient evidence from human epidemiological studies; B1--Probable Human Carcinogen, limited evidence from epidemiological studies and adequate evidence from animal studies; B2--Probable Human Carcinoagen, inadequate evidence from epidemiological studies and adequate evidence from animal studies; C-- Possible Human Carcinogen, limited evidence in animals in the absence of human data; D--Not Classified as to human carcinogenicity; and E--Evidence of Noncarcinogenicity.
(c) Inhalation RfDs in mg/m3 were converted to units of mg/kg/day assuming a 70-kg adult inhales 20 m3 of air each day.
(d) Inhalation cancer potency factors have been developed for nickel subsulfide [1.7 (mg/kg/day)-1] and nickel refinery dust [0.84 (mg/kg/day)-1]. The higher of these two values is conservatively used in this assessment.

   * = Review Pending.
   -- = Criterion has not been developed for this chemical.
 IRIS = Chemical files of the Integrated Risk Information System.
HEAST = Health Effects Assessment Summary Tables.

primarily as hyperexcitability, muscle twitching, convulsions, and depression (Borgmann et al. 1952a,b, Hayes 1982, Hodge et al. 1967, Hoogendam et al. 1962, Jager 1970). Experimental studies indicate that dogs exposed for longer periods of time to levels as low as 1 mg/kg aldrin developed hepatic and renal toxicity (Fitzhugh et al. 1964, Treon and Cleveland 1955, Walker et al. 1969). Rats fed aldrin for 2 years developed hepatic lesions and nephritis at doses of 0.5 and 50 ppm, respectively (Fitzhugh et al. 1964). Aldrin produced fetotoxic and/or teratogenic effects in hamsters fed a single oral dose of 50 mg/kg (approximately 84 ppm) and in mice fed a single oral dose of 25 mg/kg (approximately 6 ppm) (Ottolenghi et al. 1974). Aldrin produced marked effects on fertility, gestation, viability, and lactation in mice given 25 mg/kg/day in a six-generation study (Deichmann 1972). Aldrin produces chromosomal aberrations in mouse, rat, and human cells (Georgian 1974) and unscheduled DNA synthesis in rats (Probst et al. 1981) and humans (Rocchi et al. 1980). Chronic oral exposure to aldrin has produced an increase in hepatocellular tumors in mice (Davis 1965, Epstein 1975, NCI 1978). In contrast, chronic feeding studies with aldrin in rats indicate that exposure was associated with nonneoplastic changes in the liver (NCI 1978, Fitzhugh et al. 1964).

USEPA (1989) has classified aldrin as a group B2 agent (probable human carcinogen) and has developed an oral and inhalation slope factor of $1.70 \times 10^{+1}$ $(\text{mg/kg/day})^{-1}$ based on the increased incidence of liver carcinoma observed in male and female C3H mice (Davis 1965, Epstein 1975) and in male B6C3F1 mice (NCI 1978). USEPA (1989) derived an oral reference dose (RfD) for aldrin of

3.0×10^{-5} mg/kg/day based on a study in which rats were fed aldrin for 2 years and displayed liver lesions at dose levels of 0.025 mg/kg/day (0.5 ppm) and greater (Fitzhugh et al. 1964). An uncertainty factor of 1,000 was used to calculate the oral RfD.

Benzene

Benzene is readily absorbed following oral and inhalation exposure (USEPA 1985). The toxic effects of benzene in humans and other animals following exposure by inhalation include central nervous system effects, hematological effects, and immune system depression. In humans, acute exposures to high concentrations of benzene vapors have been associated with dizziness, nausea, vomiting, headache, drowsiness, narcosis, coma, and death (NAS 1976). Chronic exposure to benzene vapors can produce reduced leukocyte, platelet, and red blood cell counts (USEPA 1985). Benzene induced both solid tumors and leukemias in rats exposed by gavage (Maltoni et al. 1985). Many studies have also described a causal relationship between exposure to benzene by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC 1982, Rinsky et al. 1981, Ott et al. 1978, Wong et al. 1983).

Applying USEPA's criteria for evaluating the overall evidence of carcinogenicity to humans, benzene is classified in Group A (Human Carcinogen) based on adequate evidence of carcinogenicity from epidemiological studies. USEPA (1989) derived both an oral and an inhalation slope factor for benzene of 2.9×10^{-2} (mg/kg/day)$^{-1}$. This value was based on several studies in which

increased incidences of nonlymphocytic leukemia were observed in humans occupationally exposed to benzene principally by inhalation (Rinsky et al. 1981, Ott et al. 1978, Wong et al. 1983). USEPA (1989) is currently reviewing both oral and inhalation RfDs for benzene, for which the status is pending.

Bis(2-ethylhexyl)phthalate

Bis(2-ethylhexyl)phthalate, also known as di-ethylhexyl phthalate (DEHP), is readily absorbed following oral or inhalation exposure (USEPA 1980). Chronic exposure to relatively high concentrations of DEHP in the diet can cause retardation of growth and increased liver and kidney weights in laboratory animals (NTP 1982, USEPA 1980, Carpenter et al. 1953). Reduced fetal weight and increased number of resorptions have been observed in rats exposed orally to DEHP (USEPA 1980). DEHP is reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules following oral administration (NTP 1982).

DEHP has been classified in Group B2--Probable Human Carcinogen (USEPA 1986, 1989). USEPA (1989) calculated an oral slope factor for DEHP of 1.4×10^{-2} $(mg/kg/day)^{-1}$ based on data from the NTP (1982) study. USEPA has recommended an oral reference dose (RfD) for DEHP of 0.02 mg/kg/day based on a study by Carpenter et al. (1953) in which increased liver weight was observed in female guinea pigs exposed to 19 mg/kg bw/day in the diet for 1 year (USEPA 1989); an uncertainty factor of 1,000 was used to develop the RfD.

2-Butanone (methyl ethyl ketone)

Absorption of methyl ethyl ketone from the gastrointestinal tract and from the lungs has been inferred from systemic toxic effects observed following acute oral exposure and acute and subchronic inhalation exposures (Lande et al. 1976). Schwetz et al. (1974) reported that rats exposed to inhaled methyl ethyl ketone at concentrations of 3,000 ppm displayed retarded fetal development and teratogenic effects (acaudia, imperforate anus, and brachygnathia). In rats, subchronic exposure to 235 ppm methyl ethyl ketone vapors has resulted in fetotoxicity (Labelle and Brieger 1955). Inhaled methyl ethyl ketone also produces hepatotoxicity and neurological effects in rats (Cavender et al. 1983, Takeuchi et al. 1983, Labelle and Brieger 1955).

USEPA (1989a) determined an oral reference dose (RfD) of 5×10^{-2} mg/kg/day for methyl ethyl ketone based on a subchronic study by LaBelle and Brieger (1955) in which no adverse effects were observed in rats exposed to 235 ppm (693 mg/m^3 or 46 mg/kg/day) methyl ethyl ketone for 12 weeks. Higher doses have resulted in fetotoxic effects in rats exposed to methyl ethyl ketone via inhalation (1958 mg/m^3 or 130 mg/kg/day) (Schwetz et al. 1974). USEPA (1989b) also derived an inhalation RfD of 9×10^{-2} mg/kg/day based on the LaBelle and Brieger (1955) study in which central nervous system effects were noted in rats. An uncertainty factor of 1,000 was used to calculate both the oral and inhalation RfDs.

Butyl Benzyl Phthalate

Butyl benzyl phthalate is absorbed following oral exposure. Butyl benzyl phthalate is not especially toxic. Acute oral doses of 50,000 or 100,000 mg/kg administered to male rats resulted in testicular degeneration. Thymic atrophy was reported in both male and female rats given 100,000 mg/kg for 14 days (NTP 1982). Depressed body weight gain, testicular degeneration, and liver and kidney effects have been observed in animals subchronically administered benzyl butyl phthalate in the diet (NTP 1982, NTP 1985). Butyl benzyl phthalate has been tested for carcinogenicity in chronic feeding studies using mice and female rats, and via intraperitoneal injection in male mice (NTP 1982). In female rats, an increased incidence of myelomonocytic leukemia was observed in the high exposure group. No increased tumor incidence was noted for mice (NTP 1982).

USEPA has classified butyl benzyl phthalate in Group C--Possible Human Carcinogen. USEPA (1989) derived an oral RfD of 2×10^{-1} mg/kg/day for butyl benzyl phthalate based on a subchronic study in rats in which effects on body weight gain, testes, liver, and kidney were observed (NTP 1985). An uncertainty factor of 1,000 was used to derive the oral RfD. No inhalation criteria have been developed for butyl benzyl phthalate.

Chlorobenzene

Evidence from toxicity studies suggests that chlorobenzene is absorbed after oral, inhalation, and dermal exposure (USEPA 1985). In humans, acute and chronic exposures to chlorobenzene via inhalation or ingestion have been associated with central nervous system (CNS) effects (USEPA 1985). In animals, acute exposure by inhalation causes sensory irritation, respiratory distrees, narcosis and CNS depression which can result in death (USEPA 1985). Subchronic oral or inhalation exposure can elicit neurotoxicity, liver and kidney lesions and adverse hematological effects (USEPA 1984, USEPA 1985, Dilly 1977, Monsanto 1967, NTP 1983). Results of reproductive studies with rats and dogs also indicate that chlorobenzene induces testicular lesions (USEPA 1985).

USEPA (1989a) derived an oral chronic RfD for chlorobenzene of 2×10^{-2} mg/kg/day based on a study by Monsanto (1967) in which dogs administered chlorobenzene in capsules for 90 days exhibited liver and kidney effects; an uncertainty factor of 1,000 was used to develop the RfD. USEPA (1989b) also reported an inhalation chronic RfD for chlorobenzene of 5×10^{-3} mg/kg/day based on a study by Dilley (1977) in which rats exposed to chlorobenzene for 120 days exhibited liver and kidney effects; an uncertainty factor of 10,000 was used to develop the RfD.

Chloroform

Chloroform, a trihalomethane, is rapidly absorbed through the respiratory and gastrointestinal tracts in humans and experimental animals; dermal absorption from contact of the skin with liquid chloroform can also occur (USEPA 1985). In humans, acute exposures to chloroform may result in depression of the central nervous system, hepatic and renal damage, and death caused by ventricular fibrillation following an acute ingested dose of 10 ml (USEPA 1984). Acute exposure to chloroform may also cause irritation to the skin, eyes, and gastrointestinal tract (USEPA 1984, 1985). In experimental animals, chronic exposure may lead to fatty cyst formation in the liver (Heywood et al. 1979), renal and cardiac effects, and central nervous system depression (USEPA 1985). Chloroform has been reported to induce renal epithelial tumors in rats (Jorgenson et al. 1985) and hepatocellular carcinomas in mice (NCI 1976). Suggestive evidence from human epidemiological studies indicates that long-term exposure to chloroform and other trihalomethanes in contaminated water supplies may be associated with an increased incidence of bladder tumors (USEPA 1985). Chloroform is embryotoxic in pregnant rats and has retarded fetal development and increased the incidences of fetal resorption, acadia (absence of tail), imperforate anus, missing ribs and delayed ossification of sternebrae (Schwetz et al. 1974).

Chloroform has been classified by USEPA as a Group B2 Carcinogen (Probable Human Carcinogen) (USEPA 1989). USEPA (1989) developed an oral slope factor for chloroform of 6.1×10^{-3} $(mg/kg/day)^{-1}$ based on a study in which kidney

tumors were observed in rats exposed to chloroform in drinking water (Jorgenson et al. 1985). An inhalation slope factor of 8.1×10^{-2} $(mg/kg/day)^{-1}$ has been developed by USEPA (1989) based on an NCI (1976) bioassay in which liver tumors were observed in mice. USEPA (1989) also derived an oral reference dose (RfD) of 0.01 mg/kg/day for chloroform based on a chronic bio-assay in dogs in which liver effects were observed at 12.9 mg/kg/day (Heywood et al. 1979); an uncertainty factor of 1,000 was used to derive the RfD.

2-Chloronaphthalene

2-Chloronaphthalene appears to be absorbed following ingestion based on the detection of metabolites in the urine (Cleary et al. 1939). The extent of absorption is not known. Oral LD_{50} values for 2-chloronaphthalene in the rat and mouse are 2,078 mg/kg and 886 mg/kg, respectively (Sax 1984). The compound is reported to be moderately toxic via the oral route (Sax 1984), though no quantitative information was available on the subchronic or chronic toxicity of this chemical. It is reported that exposure to the monochloronaphthalenes are not associated with chloracne (Clayton and Clayton 1981). No health-based criteria exist for 2-chloronaphthalene.

1,2-Dichlorobenzene

1,2-Dichlorobenzene is readily absorbed through the lungs, skin, and gastrointestinal tract (USEPA 1987). The principal toxic effects of this compound in humans and experimental animals from acute and longer-term

exposure include central nervous system depression, blood dyscrasias, and lung, kidney, and liver damage (USEPA 1985, NTP 1985). Chromosome breaks also have been observed in exposed humans (USEPA 1987).

USEPA (1989b) derived an inhalation RfD of 0.04 mg/kg/day for 1,2-dichlorobenzene based on a study in which decreased body weight gain was observed in rats administered 1,2-dichlorobenzene, 7 hours/day, 5 days/week for up to 7 months (Hollingsworth et al. 1958); an uncertainty factor of 1,000 was used to derive the RfD. USEPA (1989a) also reported an oral reference dose for 1,2-dichlorobenzene of 9×10^{-2} mg/kg/day based on an NTP (1985) study in which liver effects were observed in rats exposed to 1,2-dichlorobenzene for 5 days/week for 13 weeks; an uncertainty factor of 1,000 was used to develop the RfD.

1,1-Dichloroethane

1,1-Dichloroethane (1,1-DCA) is probably less toxic than the 1,2-isomer (USEPA 1980). At one time, the compound was used as an anesthetic, but it induced cardiac arrhythmias and its use was discontinued. It is probable that human exposure to sufficiently high levels of 1,1-DCA would cause central nervous system depression and respiratory tract and skin irritation, since many of the chlorinated aliphatics cause these effects (Parker at al. 1979). However, no dose-response data concerning these effects are available. Renal damage was observed in cats exposed by inhalation in a subchronic study (Hofmann et al. 1971). Inhalation exposure of pregnant rats to high doses of 1,1-DCA

(6,000 ppm) retarded fetal development (Schwetz et al. 1974). A carcinogenicity bioassay of 1,1-DCA was limited by poor survival of both treatment and control groups, and the physical conditions of the treated animals were markedly stressed. Dose-related marginal increases in mammary gland adenocarcinomas and in hemangiosarcomas were seen in female rats, and a statistically significant increase in endometrial stromal polyps was seen in female mice; however, these data were not interpreted as providing conclusive evidence for the carcinogenicity of 1,1-DCA because of the previously mentioned limitations of the bioassay (NCI 1978).

USEPA (1989) has classified 1,1-DCA as a Group B2 agent (Probable Human Carcinogen) and reported an oral slope factor of 9.1×10^{-2} $(mg/kg/day)^{-1}$. This slope factor is based on structure-activity relationship to the isomer 1,2-dichloroethane, a Group B2 carcinogen, and on the increased incidence of hemangiosarcomas observed in rats administered 1,1-DCA via gavage (NCI 1978). USEPA (1989) developed an oral and inhalation reference dose of 0.1 mg/kg/day based on adverse renal effects seen in cats following subchronic inhalation exposure (Hofmann et al. 1971). A safety factor of 1,000 was used to develop the RfD.

1,2-Dichloroethane

Data on the toxicokinetics of 1,2-dichloroethane (1,2-DCA) in humans are limited, but data from animal studies suggest that the chemical is rapidly absorbed following oral and inhalation exposure and after dermal contact with

the liquid form of the compound (USEPA 1985). Effects of acute inhalation exposure in humans include irritation of mucous membranes in the respiratory tract and central nervous system depression (USEPA 1985). Death may occur as a result of respiratory and circulatory failure. Pathological examinations typically show congestion, degeneration, necrosis, and hemorrhagic lesions of the respiratory and gastrointestinal tracts, liver, kidney, spleen, and lungs (USEPA 1985). Adverse effects caused by less extreme exposures are generally associated with the gastrointestinal and nervous systems. Occupational exposures to 1,2-DCA vapors result in anorexia, nausea, vomiting, fatigue, nervousness, epigastric pain, irritation of the eyes and respiratory tract, and gastrointestinal, liver, and gallbladder disease (USEPA 1984, 1985). Chronic inhalation studies in animals also have revealed toxic effects including degeneration of the liver (USEPA 1985). Available data suggest that 1,2-DCA does not adversely affect reproductive or developmental processes in experimental animals except at maternally toxic levels (USEPA 1985). In long-term oral bioassays sponsored by the National Cancer Institute (NCI 1978), increased incidences of squamous-cell carcinomas of the forestomach, mammary gland adenocarcinomas, and hemangiosarcomas have been observed in rats exposed to 1,2-DCA; pulmonary adenomas, mammary adenocarcinomas, and uterine endometrial tumors have been observed in mice exposed to this chemical.

USEPA (1989) has classified 1,2-DCA in Group B2 (Probable Human Carcinogen) based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies. USEPA (1989) derived an oral and an inhalation slope factor of 9.1×10^{-2} $(mg/kg/day)^{-1}$ for

1,2-DCA based on the incidences of hemangiosarcomas in Osborne-Mendel male rats observed in the NCI (1978) gavage study.

1,1-Dichloroethene

1,1-Dichloroethene (1,1-DCE) is rapidly absorbed after oral and inhalation exposures (USEPA 1984, 1987). Humans acutely exposed to 1,1-DCE vapors exhibit central nervous system depression. In animals, the liver is the principal target of 1,1-DCE toxicity. Acute exposures result in liver damage which ranges from fatty infiltration to necrosis (USEPA 1987). Workers chronically exposed to 1,1-DCE in combination with other vinyl compounds exhibit liver dysfunction, headaches, vision problems, weakness, fatigue and neurological sensory disturbances (USEPA 1987). Chronic oral administration of 1,1-DCE to experimental animals results in both hepatic and renal toxicity (USEPA 1984, Quast et al. 1983). Inhalation or oral exposure of rats and rabbits has produced fetotoxicity and minor skeletal abnormalities, but only at maternally toxic doses. 1,1-DCE vapors produced kidney tumors and leukemia in a single study of mice exposed by inhalation, but the results of other studies were equivocal or negative (USEPA 1987, Maltoni et al. 1985).

USEPA has classified 1,1-DCE as a Group C agent (Possible Human Carcinogen) and has developed inhalation and oral slope factors of 1.2 $(mg/kg/day)^{-1}$ and 0.6 $(mg/kg/day)^{-1}$, respectively (USEPA 1985, 1989). The inhalation slope factor was based on the increased incidence of renal adenocarcinomas in male mice exposed to 1,1-DCE via inhalation for 52 weeks and observed for a total

of 121 weeks (Maltoni et al. 1985). The oral slope factor was derived by estimating an upper-limit value from negative bioassay data and assuming that a carcinogenic response occurs via ingestion, although there is no direct evidence that this is true. USEPA (1989) developed an oral reference dose (RfD) of 9×10^{-3} mg/kg/day based on the occurrence of hepatic lesions in rats chronically exposed to 1,1-DCE in drinking water (Quast et al. 1983). A safety factor of 1,000 was applied to the lowest-observed-adverse-effect level (LOAEL) of 9 mg/kg/day to derive the oral RfD.

trans-1,2-Dichloroethylene

trans-1,2-Dichloroethylene is expected to be absorbed by any route of exposure. Information on the health effects of *trans*-1,2-dichloroethylene is limited. In humans, *trans*-1,2-dichloroethylene is a central nervous system depressant, and exposure to high concentrations can result in anesthetic effects (Irish 1963). Inhalation exposure of rats to 200 ppm has been associated with pneumonic infiltration of the lungs and progressive fatty degeneration of the liver (Freundt et al. 1977). Acute exposure to higher dose levels can cause narcosis and death in rats (Torkelson and Rowe 1981). Chronic oral exposure of rats to *trans*-1,2-dichloroethylene has resulted in increased serum alkaline phosphatase (Barnes et al. 1985).

USEPA (1985) proposed a maximum contaminant level goal (MCLG) of 70 μg/liter for both *cis*- and *trans*-1,2-dichloroethylene based on the adjusted acceptable daily intake (AADI) of 350 μg/liter, assuming 20% of the exposure is via

drinking water. USEPA (1989) has derived an oral reference dose (RfD) of 2×10^{-2} mg/kg/day for *trans*-1,2-dichloroethylene based on a 90-day drinking water study conducted in mice (Barnes et al. 1985). A no-observed-adverse-effect level (NOAEL) of 17 mg/kg/day for increased serum alkaline phosphatase and an uncertainty factor of 1,000 were used to derive the RfD.

Di-n-Butyl Phthalate

Di-n-butyl phthalate is readily absorbed following oral and inhalation exposure (USEPA 1980). Acute exposures of di-n-butyl phthalate aerosol in mice have produced irritation of the eyes and upper respiratory tract mucous membranes. Extreme exposures result in labored breathing, ataxia, paresis, convulsions and death from paralysis of the respiratory system (ACGIH 1986). Workers chronically exposed to di-n-butyl phthalate in combination with other phthalate plasticizers have exhibited pain, numbness and spasms in the upper and lower extremities. Further evaluation revealed vestibular dysfunction and polyneuritis (ACGIH 1986). Reduced fetal weight, increased numbers of resorptions, and dose-related musculoskeletal abnormalities have been observed among fetuses from rats and mice exposed to very high doses of di-n-butyl phthalate during gestation (Shiota and Nishimura 1982).

USEPA (1989) calculated an oral reference dose (RfD) for di-n-butyl phthalate based on a study by Smith (1953) in which male Sprague-Dawley rats were fed a diet containing dibutyl phthalate for a period of 1 year. One-half of all rats receiving the highest dibutyl phthalate concentration (1.25% of diet, or

600 mg/kg/day) died during the first week of exposure. The remaining animals survived the study with no apparent adverse effects. Using a NOAEL of 125 mg/kg/day (0.25% dibutyl phthalate in diet) and an uncertainty factor of 1,000, an oral reference dose (RfD) of 0.1 mg/kg/day was derived; a LOAEL of 600 mg/kg/day (1.25% dibutyl phthalate in diet) was observed in this study.

Di-n-Octyl Phthalate

Di-*n*-octyl phthalate is not especially toxic. It is a severe eye and a mild skin irritant in rabbits (NIOSH 1985, NTP/IRLG 1982, USEPA 1980). Fetotoxicity and developmental abnormalities were observed in the offspring of rats administered 5 g/kg intraperitoneal injections on days 6 to 15 of gestation (NTP/IRLG 1982, USEPA 1980). No health-based criteria have been developed for this compound by USEPA.

Dieldrin

Dieldrin can be absorbed by humans from the gastrointestinal tract following ingestion of the pesticide (NIOSH 1978), and absorbed through human skin following percutaneous exposure (Feldmann and Maibach 1974). NIOSH (1978) reported that another possible route of absorption by humans is through inhalation (NIOSH 1978). Reported effects in humans following acute exposure to dieldrin include malaise, incoordination, headache, dizziness, gastrointestinal disturbances, and major motor convulsions (NRC 1982). Dieldrin is acutely toxic to laboratory animals by the oral, dermal, and

inhalation routes. It is mildly irritating to the eye and skin. Dieldrin

affects the central nervous system, producing irritability, tremors, and

convulsions (Health and Vandekar 1964). In experimental animals chronic oral

administration of dieldrin is associated with liver and kidney damage (Walker

et al. 1969, Treon and Cleveland 1955, Murphy and Korschgen 1970). Oral

administration of dieldrin is reported to result in reproductive toxicity,

fetotoxicity, and teratogenicity in mice and hamsters (Diechmann 1972,

Ottolenghi et al. 1974). Dieldrin is reported to cause a significant

dose-related increase in the incidence of hepatocellular carcinoma in mice

exposed in the diet (NCI 1978, Davis and Fitzhugh 1962).

USEPA has classified dieldrin in Group B2--Probable Human Carcinogen based on

inadequate evidence of carcinogenicity from human studies and sufficient

evidence of carcinogenicity from animal studies (USEPA 1989). USEPA (1989)

reported a slope factor of 1.6×10^1 (mg/kg/day)$^{-1}$ for both oral and inhalation

exposures based on several studies in which hepatocellular carcinomas were

observed in mice administered dieldrin in the diet (Walker et al. 1972, Thorpe

and Walker 1973, NCI 1978, Tennekes et al. 1981). USEPA (1989) has

established an oral reference dose (RfD) of 5.0×10^{-5} mg/kg/day for dieldrin

based on liver lesions observed in rats (Walker et al. 1969). The RfD was

derived using a no-observed-effect level (NOEL) of 0.005 mg/kg/day and an

uncertainty factor of 100.

2,4-Dimethylphenol

Little information is available on the health effects from exposure to 2,4-dimethylphenol (2,4-DMP). Exposure to 2,4-DMP is usually as a component of a complex mixture (USEPA 1980). It is readily absorbed through the skin of animals and has been reported to be an adenosine triphosphate (ATP) blocking agent (USEPA 1980). In rats, dermal and oral LD_{50}s for 2,4-dimethylphenol of 1,040 mg/kg and 3,200 mg/kg have been reported (Uzhdovini et al. 1974). Also, 2,4-dimethlyphenol has been reported to act as a cancer promoting agent in skin painting studies in rats (Boutwell & Bosch 1959). No health-based criteria have been developed for this compound by USEPA.

Ethylbenzene

Ethylbenzene is absorbed via inhalation and distributed throughout the body in rats; the highest levels were detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al. 1980). In humans, short-term inhalation exposure to 435 mg/m^3 ethylbenzene for 8 hours can result in sleepiness, fatigue, headache, and mild eye and respiratory irritation (Bardodej and Bardodejova 1970); eye irritation has also been observed in experimental animals exposed to ethylbenzene (USEPA 1987). Increased weights and cloudy swelling were observed in the liver and kidney of rats exposed to ethylbenzene by gavage at a dose of 408 mg/kg/day for 182 days (Wolf et al. 1956). A single oral dose of ethylbenzene administered to male and female Wistar-derived rats was reported to have an LD_{50} of 3,500 mg/kg body weight,

with systemic effects occurring primarily in the liver and kidney (Wolf et al. 1956). Maternal toxicity was observed in rats exposed by inhalation to 4,348 mg/m^3 ethylbenzene for 6-7 hours/day during the first 19 days of gestation (Hardin et al. 1981).

USEPA (1989) derived an oral reference dose of 0.1 mg/kg/day for ethylbenzene based on the chronic study by Wolf et al. (1956) in which no liver or kidney effects were observed in rats exposed to 136 mg/kg/day. An uncertainty factor of 1,000 was applied to the no-observed-effect-level to derive the reference dose.

Isophorone

Isophorone is absorbed following oral administration in animals (Rohm and Haas 1972, NTP 1986), and nearly 93% is excreted in the urine within 24 hours (Strasser 1988). Absorption in the lungs can be inferred from the systemic toxicity observed in animals following inhalation exposure. Humans acutely exposed to isophorone vapors as high as 400 ppm experienced eye, nose, and throat irritation, nausea, faintness, headaches, dizziness, and narcosis; however, complaints of irritation and narcosis decreased at concentrations of 40 and 85 ppm (ACGIH 1986). Acute inhalation exposure in animals results in respiratory tract irritation, slight lung congestion, and central nervous system (CNS) depression (De Ceaurriz et al. 1981a,b; Hazleton Labs 1964). Acute oral administration of isophorone to rats has produced CNS depression, ptosis, absence of righting reflex, and prostration (Hazleton Labs 1964).

Isophorone is a weak dermal irritant to rabbits and guinea pigs following acute exposures (Truhaut et al. 1972). Rats chronically exposed to isophorone by oral gavage have exhibited hyperkerotosis of the forestomach, hepatic coagulative necrosis and cytomegaly, and inflammation, tubular cell hyperplasia, and tubular mineralization of the kidney (NTP 1984, USEPA 1986). Isophorone is not teratogenic or fetotoxic in mice or rats at concentrations up to 115 ppm (Biodynamics 1984). Increased incidences of renal tubular cell adenomas and adenocarcinomas and preputial gland carcinomas have been observed in rats following chronic oral exposures (NTP 1986).

USEPA (1989b) has classified isophorone as a group C agent (possible human carcinogen) and has developed an oral slope factor of 4.1×10^{-3} (mg/kg/day)$^{-1}$ based on the increased incidence of kidney and preputial gland tumors observed in rats (NTP 1986, USEPA 1986). USEPA (1989a) derived an oral reference dose (RfD) for isophorone of 2.0×10^{-1} mg/kg/day based on a study in which dogs administered isophorone in capsules for 90 days displayed no treatment-related changes in hematology, blood chemistry, organ weight, urinalysis, gross appearance or cellular changes in liver or kidney at doses of 150 mg/kg/day (Rohm and Haas 1972). However, a chronic study conducted in rats noted kidney lesions (tubular hyperplasia and mineralization) at higher doses of 179 mg/kg/day (NTP 1984). An uncertainty factor of 1,000 was used to calculate the oral RfD. USEPA has not derived an inhalation RfD for isophorone. ACGIH (1986) reports a ceiling limit of 5 ppm.

Methylene Chloride

Methylene chloride is absorbed following oral and inhalation exposure. The amount of airborne methylene chloride absorbed following inhalation exposure increases in direct proportion to its concentration in inspired air, the duration of exposure, and physical activity. Dermal absorption has not been accurately measured (USEPA 1985a). Acute human exposure to methylene chloride may result in irritation of eyes, skin, and respiratory tract; central nervous system depression; elevated carboxyhemoglobin levels; and circulatory disorders that may be fatal (USEPA 1980). Chronic exposure of animals can produce renal and hepatic toxicity (NCA 1982). Methylene chloride is mutagenic for *Salmonella typhimurium* and produces mitotic recombination in yeast (USEPA 1989a). Several inhalation studies conducted in animals provide clear evidence of methylene chloride's carcinogenicity (NTP 1986). There is only suggestive evidence in experimental animals that hepatocellular carcinomas and neoplastic nodules arise from oral exposure (USEPA 1985a,b).

USEPA (1989a) classified methylene chloride in Group B2--Probable Human Carcinogen. It has been concluded by USEPA (1985b) that the induction of distant site tumors from inhalation exposure and the borderline significance for induction of tumors in a drinking water study are an adequate basis for concluding that methylene chloride be considered a probable human carcinogen via ingestion as well as inhalation. USEPA (1989a) derived an inhalation slope factor of 1.4×10^{-2} $(\text{mg/kg/day})^{-1}$ based on the results of a National Toxicology Program (NTP) inhalation bioassay conducted in rats and mice (NTP

1986). Mammary tumors were noted in rats, while lung and liver tumors were observed in mice. USEPA (1989a) determined an oral slope factor of 7.5×10^{-3} $(mg/kg/day)^{-1}$ based on the results of the NTP (1986) inhalation bioassay and on an ingestion bioassay conducted by the National Coffee Association (NCA 1983). In the NCA study, hepatocellular adenomas and/or carcinomas were observed in male mice. An oral reference dose (RfD) of 0.06 mg/kg/day has been developed by USEPA (1989a) based on a 2-year rat drinking water bioassay (NCA 1982) that identified no-observed-effect levels (NOELs) of 5.85 and 6.47 mg/kg/day for male and female rats, respectively. Liver toxicity was observed at doses of 52.58 and 58.32 mg/kg/day for males and females, respectively. An uncertainty factor of 100 was used to derive the RfD. USEPA (1989b) has established an inhalation RfD of 3 mg/m^3 based on a study by Nitschke et al. (1988) in which rats were exposed to 200 ppm (694.8 mg/m^3) for 2 years. A safety factor of 100 was used to derive the RfD. This RfD is currently undergoing verification by USEPA (1989a).

Naphthalene

Naphthalene is rapidly absorbed when inhaled but is more slowly absorbed by ingestion or through the skin (USEPA 1982). Inhalation and oral exposure to naphthalene may cause nausea, headaches, vertigo, vomiting, abdominal pain, and liver and kidney damage in both humans and experimental animals (Linick 1983, Ojwang et al. 1985, Gidron and Leurer 1956, Gupta et al. 1979, Kurz 1987, Rao and Pandya 1981). Acute hemolytic anemia are reportedly caused by ingestion or inhalation of relatively large quantities of naphthalene (USEPA

1982, Valaes et al. 1963). Optical neuritis, injuries to the cornea, and opacities of the lens also may result after inhalation exposure or ingestion (USEPA 1982). Naphthalene is a mild eye irritant in rabbits, and cataracts can be induced after oral administration (USEPA 1982). Application to the skin produces erythema and slight edema in rabbits (USEPA 1982). Retarded cranial ossification and heart development are reported among offspring of rats injected intraperitoneally with naphthalene on gestational days 1 to 15 (USEPA 1982). A significant reduction in the average number of live pups per litter was reported following a single oral dose of naphthalene (Plasterer et al. 1985). There are no epidemiologic or case studies available suggesting that naphthalene is carcinogenic in humans (USEPA 1984). This compound is not generally considered to be carcinogenic in experimental animals (USEPA 1984).

USEPA (1989) developed an oral reference dose of 0.4 mg/kg/day for naphthalene based on the development of ocular and systemic lesions in rats (Schmahl 1955, USEPA 1986) and occupational data on coke-oven workers. An uncertainty factor of 100 was applied to the animal data in the development of the reference dose.

Nitrobenzene

Nitrobenzene is absorbed by all possible routes, but absorption primarily occurs through the respiratory tract and skin (USEPA 1980); approximately 80% of inhaled nitrobenzene is absorbed (USEPA 1980). In humans long-term occupational exposure to nitrobenzene can result in cyanosis,

methemoglobinemia, jaundice, anemia, sulfhemoglobinemia, and dark urine (USEPA 1980). Short-term exposure to high levels of nitrobenzene can result in cyanosis, and if severe, the individual can go into a coma (Piotrowski 1967). Hematologic, adrenal, renal, and hepatic lesions have been reported in rats and mice exposed to nitrobenzene in air for 90 days (CIIT 1984). There is also limited evidence that exposure to nitrobenzene can result in changes in the tissues of the chorion and placenta in pregnant women (Dorigan and Hushon 1976); menstrual disturbances after chronic nitrobenzene exposure have also been reported (USEPA 1980).

USEPA (1989a) developed an inhalation RfD for nitrobenzene of 6×10^{-4} mg/kg/day based on a study in which hematological, adrenal, renal, and hepatic lesions were observed in mice following inhalation exposure to nitrobenzene (CIIT 1984) and using an uncertainty factor of 10,000. USEPA (1989b) also developed an oral RfD for nitrobenzene of 5×10^{-4} mg/kg/day based on the CIIT study based on route-to-route extrapolation and using an uncertainty factor of 10,000 (USEPA 1989b). USEPA (1989b) is currently evaluating the carcinogenic potential of nitrobenzene.

Phenol

Phenol is readily absorbed through the gut, by inhalation, and percutaneously (USEPA 1980). Signs of acute phenol toxicity in humans and experimental animals are central nervous system depression, collapse, coma, cardiac arrest, and death. Acutely toxic doses can also cause extensive necrosis at the site

of exposure (eyes, skin, oropharynx) (USEPA 1980). In experimental animals subchronic oral and inhalation studies suggest that kidney, pulmonary, myocardial, and liver damage are associated with exposure, although many of these studies were poorly designed (USEPA 1980, 1984). Oral administration of phenol to pregnant rats during gestational days 6 to 15 resulted in a significant reduction in fetal body weight (NTP 1983). Phenol exhibited tumor-promoting activity in the mouse skin painting system following initiation with 9,10-dimethyl-1,2-benzanthracene (DMBA) or benzo[a]pyrene (B[a]P), and it exhibited cutaneous carcinogenic activity in a sensitive mouse strain when applied at concentrations that produced repeated skin damage (USEPA 1980).

USEPA (1989) has established an oral reference dose (RfD) of 0.6 mg/kg/day for phenol based on reduced fetal body weight in rats (NTP 1983). A no-observed-adverse-effect level (NOAEL) of 60 mg/kg/day and a safety factor of 100 were used to derive RfD. USEPA has not yet established an inhalation RfD (USEPA 1989).

Polychlorinated Biphenyls (PCBs)

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

PCBs are readily absorbed through the gastrointestinal tract and somewhat less readily through the skin; PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (USEPA 1985). Dermatitis and chloracne (a disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a study of women who had consumed PCB-contaminated fish from Lake Michigan (USEPA 1985). Reproductive, hepatic, immunotoxic, and immunosuppressive effects appear to be the most sensitive end points of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents (USEPA 1985). A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors including liver tumors in animals exposed to the mixtures for long periods (Kimbrough et al. 1975, NCI 1978, Schaeffer et al. 1984, Norback and Weltman 1985). Studies have suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (USEPA 1985).

USEPA (1984, 1989) classified PCB as a Group B2 agent (Probable Human Carcinogen) based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. The USEPA Carcinogen Assessment Group (USEPA 1989) calculated an oral slope factor of 7.7 $(mg/kg/day)^{-1}$ for PCBs based on

the incidence of hepatocellular carcinomas and adenocarcinomas in female Sprague-Dawley rats exposed to a diet containing Aroclor 1260 as reported in a study by Norback and Weltman (1985). Clement Associates has derived an oral RfD of 1×10^{-4} mg/kg/day for Aroclor 1016 based on a chronic oral study conducted in monkeys (Barsotti and Van Miller 1984). A no-observed-adverse-effect level of 0.25 ppm (0.01 mg/kg/day) for fetotoxicity was identified from this study. A safety factor of 100 (10 to account for interspecies extrapolation and 10 to account for the variation among the members of the human population) was used to calculate the RfD.

Polycyclic Aromatic Hydrocarbons (Carcinogenic)

PAHs occur in the environment as complex mixtures containing numerous PAHs of varying carcinogenic potencies. Only a few components of these mixtures have been adequately characterized, and only limited information is available on the relative potencies of different compounds.

PAH absorption following oral and inhalation exposure is inferred from the demonstrated toxicity of PAHs following ingestion and inhalation, respectively (USEPA 1984a). PAHs are also absorbed following dermal exposure (Kao et al. 1985). It has been suggested that simultaneous exposure to carcinogenic PAHs such as benzo[a]pyrene and particulate matter can increase the effective dose of the compound (ATSDR 1987). Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity; the primary effect is dermatitis (NIOSH 1977). PAHs have also been shown to cause cytotoxicity

in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato et al. 1981). Destruction of the sebaceous glands, hyperkeratosis, hyperplasia, and ulceration have been observed in mouse skin following dermal application of the carcinogenic PAHs (Santodonato et al. 1981). The carcinogenic PAHs have also been shown to have an immunosuppressive effect in animals (ATSDR 1987). Nonneoplastic lesions have been observed in animals exposed to the more potent carcinogenic PAHs but only after exposure to levels well above those required to elicit a carcinogenic response. Carcinogenic PAHs are believed to induce tumors both at the site of application and systemically. Neal and Rigdon (1967) reported that oral administration of 250 ppm benzo[a]pyrene for approximately 110 days led to forestomach tumors in mice. Thyssen et al. (1981) observed respiratory tract tumors in hamsters exposed to up to 9.5 mg/m^3 benzo[a]pyrene for up to 96 weeks.

Benzo[a]pyrene is representative of the carcinogenic PAHs and is classified by USEPA in Group B2--Probable Human Carcinogen--based on sufficient evidence of carcinogenicity from animal studies and inadequate evidence from epidemiological studies (USEPA 1984b, 1989). USEPA (1984b) calculated an oral slope factor of 11.5 (mg/kg/day)$^{-1}$ for carcinogenic PAHs (specifically benzo[a]pyrene) based on the study by Neal and Rigdon (1967).

Polycyclic Aromatic Hydrocarbons (Noncarcinogenic)

Polycyclic aromatic hydrocarbons (PAHs) occur in the environment as complex mixtures of which only a few components have been adequately characterized. Only limited information is available on the relative potencies of the "noncarcinogenic" PAHs. However, many have been shown to have some weak carcinogenic activity, or to act as promoters or cocarcinogens.

PAH absorption following oral and inhalation exposure is inferred from the demonstrated toxicity of PAHs following these routes of administration (USEPA 1984). PAHs are also absorbed following dermal exposure (Kao et al. 1985). Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity; the primary effect is dermatitis (NIOSH 1977). PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato et al. 1981). Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity but these effects are generally seen at high doses (Santodonato et al. 1981). Slight morphological changes in the liver and kidney of rats have been reported following oral exposure to acenaphthene for 40 days (USEPA 1984). Subchronic oral administration of naphthalene to rabbits and rats has resulted in cataract formation (Schmahl 1955).

USEPA (1989) developed an oral reference dose of 0.4 mg/kg/day for naphthalene based on the development of ocular and systemic lesions in rats (Schmahl 1955,

USEPA 1986). An uncertainty factor of 100 was applied to the animal data in the development of the reference dose.

1,1,2,2-Tetrachloroethane

In humans, absorption of a single inhalation dose of 1,1,2,2,-tetrachloro-ethane vapor was reported to be 97%; absorption of this chemical from the gastrointestinal tract is inferred from studies in which an increased incidence of liver tumors was reported in mice exposed in the diet (USEPA 1984). The effects associated with occupational exposure to 1,1,2,2-tetrachloroethane by inhalation or dermal routes are primarily neurological and include, tremors, headache, numbness, excessive perspiration, and anorexia (USEPA 1984). In experimental animals, subchronic inhalation exposure to 1,1,2,2-tetrachloroethane is associated with liver effects, decreased hemoglobin content of red blood cells, decreased hematocrit, and fluctuations in white blood cell count (Schmidt et al. 1972, Navrotskiy et al. 1971, Horiuchi et al. 1962). 1,1,2,2-Tetrachloroethane is a liver carcinogen when administered orally to mice (NCI 1978).

USEPA (1989) classified 1,1,2,2-tetrachloroethane in Group C--Possible Human Carcinogen based on increased incidence of hepatocellular carcinoma in mice. USEPA (1989) developed an oral slope factor of 0.2 $(mg/kg/day)^{-1}$ based on the study conducted by NCI (1978) in which a highly significant dose-related increase in the incidence of hepatocellular carcinomas was observed in both male and female mice. An inhalation slope factor of 0.2 $(mg/kg/day)^{-1}$ was

also calculated from these data (USEPA 1989). USEPA (1987) also has derived
an interim oral reference dose (RfD) of $4.6x10^{-4}$ mg/kg/day for 1,1,2,2-
tetrachloroethane based on an inhalation study by Schmidt (1972) in which rats
were exposed to 1,1,2,2-tetrachloroethane vapor for 4 hours/day for 265 days.
In this study, decreased body weight, increased white blood cell count, and
increased hepatic fat content were observed. Using a LOAEL of 0.456 mg/kg/day
and applying an uncertainty factor of 1,000, the interim RfD was derived.

Tetrachloroethylene

Tetrachloroethylene is absorbed following inhalation (IARC 1979) and oral
(USEPA 1985a,b) exposure. Tetrachloroethylene vapors and liquid also can be
absorbed through the skin (USEPA 1985a,b). The principal toxic effects of
tetrachloroethylene in humans and animals following acute and longer-term
exposures include central nervous system (CNS) depression and fatty
infiltration of the liver and kidney with concomitant changes in serum enzyme
activity levels indicative of tissue damage (USEPA 1985a,b, Buben and
O'Flaherty 1985). Humans exposed to doses of between 136 and 1,018 mg/m^3 for
5 weeks develop central nervous system effects, such as lassitude and signs of
inebriation (Stewart et al. 1974). The offspring of female rats and mice
exposed to high concentrations of tetrachloroethylene for 7 hours daily on
days 6-15 of gestation developed toxic effects, including a decrease in fetal
body weight in mice and a small but significant increase in fetal resorption
in rats (Schwetz et al. 1975). Mice also exhibited developmental effects,
including subcutaneous edema and delayed ossification of skull bones and

sternebrae (Schwetz et al. 1975). In a National Cancer Institute bioassay (NCI 1977), increased incidences of hepatocellular carcinoma were observed in both sexes of B6C3F1 mice administered tetrachloroethylene in corn oil by gavage for 78 weeks. Increased incidences of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have also been observed in long term bioassays in which rats were exposed to tetrachloroethylene by inhalation (NTP 1986).

USEPA (1989b) classifies tetrachloroethylene as a Group B2 carcinogen (Probable Human Carcinogen). USEPA (1989b, 1985b) has derived an oral slope factor of 5.1×10^{-2} $(mg/kg/day)^{-1}$ based on liver tumors observed in the NCI (1977) gavage bioassay for mice. The inhalation slope factor for tetrachloroethylene of 3.3×10^{-3} $(mg/kg/day)^{-1}$ is based on an NTP (1986) bio-assay in rats and mice in which leukemia and liver tumors were observed (USEPA 1989b). Both slope factors are currently under review by USEPA (1989a). USEPA (1989a,b) also has derived an oral reference dose (RfD) of 1×10^{-2} mg/kg/day for tetrachloroethylene based on a 6-week gavage study by Buben and O'Flaherty (1985). In this study, liver weight/body weight ratios were significantly increased in mice and rats treated with 71 mg/kg/day tetrachloroethylene but not in animals treated with 14 mg/kg/day. Using a NOAEL of 14 mg/kg/day and applying an uncertainty factor of 1,000, the RfD was derived.

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Toluene

Toluene is absorbed in humans following both inhalation and dermal exposure (USEPA 1985). In humans, the primary acute effects of toluene vapor are central nervous system (CNS) depression and narcosis. These effects occur at concentrations of 200 ppm (754 mg/m^3) (von Oettingen et al. 1942a,b). In experimental animals, acute oral and inhalation exposures to toluene can result in central nervous system (CNS) depression and lesions of the lungs, liver, and kidneys (USEPA 1987). The earliest observable sign of acute oral toxicity in animals is depression of the CNS, which becomes evident at approximately 2,000 mg/kg (Kimura et al. 1971). In humans, chronic exposure to toluene vapors at concentrations of approximately 200 and 800 ppm has been associated with CNS and peripheral nervous system effects, hepatomegaly, and hepatic and renal function changes (USEPA 1987, Anderson et al. 1983). Toxic effects following prolonged exposure of experimental animals to toluene are similar to those seen following acute exposure (Hanninen et al. 1976, von Oettingen et al. 1942a). In rats, chronic exposure to toluene via inhalation results in CNS toxicity and a dose-related reduction in hematocrit values (CIIT 1980). There is some evidence in mice that oral exposure to greater than 0.3 ml/kg toluene during gestation results in embryotoxicity (Nawrot and Staples 1979). Inhalation exposure of up to 1,000 mg/m^3 by pregnant rats during gestation has been associated with significant increases in skeletal retardation (Hudak and Ungvary 1978).

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USEPA (1989a) has derived an oral risk reference dose (RfD) of 0.3 mg/kg/day

for toluene based on a 24-month inhalation study in which rats were exposed to

concentrations as high as 300 ppm (29 mg/kg/day) and hematological parameters

were examined (CIIT 1980). No adverse effects were observed in any of the

treated animals. Using a no-observed-adverse-effect level (NOAEL) of

29 mg/kg/day and an uncertainty factor of 100, the oral RfD was derived.

USEPA (1989b) reported an inhalation RfD for toluene of 2 mg/m^3 based on the

development of adverse CNS effects in humans (Anderson et al. 1983). An

uncertainty factor of 100 was used.

1,1,1-Trichloroethane

Like other chlorinated aliphatic hydrocarbons, 1,1,1-trichloroethane

(1,1,1-TCA, methyl chloroform) is rapidly and completely absorbed following

both oral and inhalation exposure. Pulmonary absorption is initially large

and gradually decreases to a steady-state condition. Absorption through the

skin is slow. 1,1,1-TCA distributes throughout the body and readily crosses

the blood-brain barrier (USEPA 1984). The most notable toxic effects of

1,1,1-TCA inhalation exposure in humans and animals are central nervous system

depression, including anesthesia at very high concentrations, and impairment

of coordination, equilibrium, and judgment at lower concentrations (350 ppm

and above). In both humans and animals, cardiovascular effects, including

premature ventricular contractions, decreased blood pressure, and

sensitization to epinephrine-induced arrhythmia can result from acute exposure

to high concentrations of 1,1,1-TCA vapor (USEPA 1985). Fatty liver changes

have been reported in guinea pigs following subchronic inhalation exposure (Torkelson et al. 1958). NTP (1984) reported preliminary results of bioassays in rats and mice indicating that oral administration of 1,1,1-TCA increases the incidence of hepatocellular carcinomas in female mice but not for male rats. This study was inadequate to evaluate the carcinogenicity of 1,1,1-TCA in female rats and male mice.

USEPA (1989a) calculated an oral reference dose (RfD) for 1,1,1-trichloroethane based on an inhalation study by Torkelson et al. (1958) in which rats, rabbits, guinea pigs and monkeys were exposed to 1,1,1-TCA vapor. A no-observed-adverse-effect (NOAEL) of 500 ppm (2,730 mg/m^3, or 90 mg/kg/day) was identified from this study. Using the NOAEL of 90 mg/kg/day and an uncertainty factor of 1,000, a RfD of 9×10^{-2} mg/kg/day was derived. An inhalation RfD of 0.3 mg/kg/day for 1,1,1-TCA also has been determined by USEPA (1989b) based on this same study, in which hepatotoxicity was observed in guinea pigs. An uncertainty factor of 1,000 was used in calculating the RfD.

Trichloroethylene

Absorption of trichloroethylene (TCE) from the gastrointestinal tract is virtually complete. Absorption following inhalation exposure is proportional to concentration and duration of exposure (USEPA 1985). TCE is a central nervous system depressant following acute and chronic exposures. In humans, single oral doses of 15 to 25 ml (21 to 35 grams) of TCE have resulted in

vomiting and abdominal pain, followed by transient unconsciousness (Stephens 1945). High-level exposure can result in death due to respiratory and cardiac failure (USEPA 1985). Hepatotoxicity has been reported in human and animal studies following acute exposure to TCE (USEPA 1985). Nephrotoxicity has been observed in animals following acute exposure to TCE vapors (ACGIH 1986, Torkelson and Rowe 1981). Subacute inhalation exposures of mice have resulted in transient trichloroethylene-induced increased liver weights (Kjellstrand et al. 1983). Industrial use of TCE is often associated with adverse dermatological effects including reddening and skin burns on contact with the liquid form, and dermatitis resulting from vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported after exposure to TCE in dilute, aqueous solutions (USEPA 1985). Trichloroethylene has caused significant increases in the incidence of hepatocellular carcinomas in mice (NCI 1976) and renal tubular-cell neoplasms in rats exposed by gavage (NTP 1983), and pulmonary adenocarcinomas in mice following inhalation exposure (Fukuda et al. 1983, Maltoni et al. 1986). Trichloroethylene was mutagenic in *Salmonella typhimurium* and in *E. coli* (strain K-12), utilizing liver microsomes for activation (Greim et al. 1977).

USEPA (1989a) classified trichloroethylene in Group B2--Probable Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animals studies. An oral slope factor of 1.1×10^{-2} $(mg/kg/day)^{-1}$ has been derived by USEPA (1989) based on two gavage studies conducted in mice in which an increased incidence of liver tumors were observed (Maltoni et al. 1986, Fukuda et al. 1983). An inhalation slope

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factor of 1.7×10^{-2} (mg/kg/day)$^{-1}$ has been derived for trichloroethylene (USEPA 1989) based on an increased incidence of lung tumors in mice (NCI 1976). USEPA (1987) developed an oral reference dose (RfD) of 7.35×10^{-3} mg/kg/day based on a subchronic inhalation study in rats in which elevated liver weights were observed following exposure to 55 ppm, 5 days/week for 14 weeks (Kimmerle and Eben 1973). A safety factor of 1,000 was used to calculate the RfD. However, this RfD is currently under review by USEPA.

Vinyl Chloride

Vinyl chloride is rapidly absorbed in rats following oral and inhalation exposure, while dermal absorption of vinyl chloride is minor (USEPA 1985). At high inhalation exposure levels, workers have experienced dizziness, head-aches, euphoria, and narcosis. In experimental animals, inhalation exposure to high levels of vinyl chloride can induce narcosis and death. Lower doses result in ataxia, narcosis, congestion and edema of the lungs, and hyperemia in the liver (USEPA 1985). Chronic inhalation exposure of workers to vinyl chloride is associated with hepatotoxicity, central nervous system disturbances, pulmonary insufficiency, cardiovascular toxicity, gastrointestinal toxicity, and acro-osteolysis (USEPA 1985). Experimental animals chronically exposed via inhalation or ingestion have exhibited effects involving the liver, spleen, kidneys, hematopoietic system, and skeletal system (USEPA 1984). Feron et al. (1975) found that administration of vinyl chloride to rats by gavage resulted in hematologic, biochemical, and organ-weight effects at doses above 30 mg/kg/day. Evidence for an association

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between human exposure to vinyl chloride and birth defects or fetal loss is conflicting (USEPA 1987). Human exposure to vinyl chloride has been associated with an increased incidence of hepatic angiosarcoma and brain, lung, and hemolymphopoietic cancers. In animal studies, chronic inhalation and ingestion of vinyl chloride at levels as low as 1.7 and 5 mg/kg/day have induced cancer in the liver and in other tissues of rats and mice (IARC 1979; Feron et al. 1981; Maltoni et al. 1980, 1981).

USEPA (1989) has classified vinyl chloride in Group A (Human Carcinogen) based on adequate evidence of carcinogenicity from epidemiological studies. USEPA (1989) reported an oral slope factor of 2.3 $(mg/kg/day)^{-1}$ for vinyl chloride based on the long-term ingestion study in rats in which lung tumors were observed (Feron et al. 1981). The inhalation slope factor for vinyl chloride is 2.95×10^{-1} $(mg/kg/day)^{-1}$ (USEPA 1989) and is based on a chronic inhalation study conducted by Maltoni et al. (1980, 1981) in which liver tumors were observed in rats.

Xylenes

The three xylene isomers, compounds that have the same chemical constituents in a different configuration, have similar toxicological properties and are discussed together. Data from animals and humans suggest that approximately 60% of an inhaled dose is absorbed. Inference from metabolism and excretion studies suggests that absorption of orally administered xylene is nearly complete. Dermal absorption is reported to be minor following exposure to

xylene vapor but may be significant following contact with the liquid (USEPA 1985). In humans, acute inhalation exposures to relatively high concentrations of xylene adversely affect the central nervous system and lungs and can irritate mucous membranes (USEPA 1987, Hake et al. 1981). Savolainen et al. (1980) observed that body balance and manual coordination were impaired in eight male students following inhalation exposure to m-xylene. However, tolerance against the observed effects developed during one work week. In experimental rats, long-term inhalation exposure to o-xylene resulted in hepatomegaly (Tatrai et al. 1981). Oral exposure to 200 mg/kg xylene in the diet for up to 6 months was also associated with liver toxicity, specifically the development of intracellular vesicles (Bowers et al. 1982). Prolonged oral exposures in mice resulted in hyperactivity, a manifestation of CNS toxicity (NTP 1986). Xylene appears to be fetotoxic and may increase the incidence of visceral and skeletal malformations in offspring of exposed experimental animals (Mirkova et al. 1983). There is suggestive evidence that xylene is carcinogenic in experimental animals when exposed by oral gavage (Maltoni et al. 1985).

USEPA (1989a) calculated an oral reference dose (RfD) for mixed xylenes of 2 mg/kg/day based on an NTP (1986) study in which male rats given a gavage dose of 179 mg/kg/day for 103 weeks did not exhibit hyperactivity, decreased body weight or a significant increased mortality. The oral RfD was derived using the no-observed-adverse-effect level (NOAEL) of 179 mg/kg/day and an uncertainty factor of 100. USEPA (1989b) reported an inhalation RfD for mixed xylenes of 0.3 mg/m^3 based on a study in which CNS effects, and nose and

throat irritation were observed in humans exposed to 20 ppm (27 mg/m^3) for 5 days (Hake et al. 1981, Carpenter et al. 1975); an uncertainty factor of 100 was used to develop the RfD.

4.2.2 Inorganic Chemicals

Antimony

Antimony is a metal which occurs both in the trivalent and pentavalent oxidation states (USEPA 1980). Absorption of this metal via oral and inhalation routes of exposure is low (USEPA 1980). Humans and animals exposed acutely by oral or inhalation exposures to either trivalent or pentavalent forms of antimony displayed electrocardiogram (ECG) changes and myocardial lesions (USEPA 1980). Pneumoconiosis has been observed in humans exposed by acute inhalation and dermatitis has occurred in individuals exposed either orally or dermally. Oral administration of therapeutic doses in humans has been associated with nausea, vomiting, and hepatic necrosis (USEPA 1980). Chronic exposure by inhalation of antimony has led to respiratory effects including macrophage proliferation and activity, fibrosis and pneumonia in animals (USEPA 1980). Chronic oral exposure in rats has resulted in altered blood glucose and blood cholesterol levels and decreased lifespan (Schroeder et al. 1970). A single report (Balyeava 1967) noted an increase in spontaneous abortions, premature births, and gynecological problems in 318 female workers exposed to a mixture of antimony metal, antimony trioxide, and antimony pentasulfide dusts.

USEPA (1989) derived an oral reference dose (RfD) of 4×10^{-4} mg/kg/day for antimony based on a chronic oral study (Schroeder et al. 1970) in which rats given the metal in drinking water had altered blood glucose and blood cholesterol levels and decreased lifespan. An uncertainty factor of 1,000 and a LOAEL of 0.35 mg/kg/day were used to derive the oral RfD.

Arsenic

Both inorganic and organic forms of arsenic are readily absorbed via the oral and inhalation routes. Soluble forms are more readily absorbed than insoluble forms (USEPA 1984). Approximately 95% of soluble inorganic arsenic administered to rats is absorbed from the gastrointestinal tract (Coulson et al. 1935, Ray-Bettley and O'Shea 1975). Approximately 70%-80% of arsenic deposited in the respiratory tract of humans has been shown to be absorbed (Holland et al. 1959). Dermal absorption is not significant (USEPA 1984). Acute exposure of humans to metallic arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy (USEPA 1984). Chronic exposure of humans to this metal can produce toxic effects on both the peripheral and central nervous systems, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular damage (USEPA 1984, Tseng 1977). Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (USEPA 1984). Arsenic is a known human carcinogen. Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown that inhalation of arsenic is strongly associated with

lung cancer and perhaps with hepatic angiosarcoma (USEPA 1984). Ingestion of
arsenic has been linked to a form of skin cancer and more recently to bladder,
liver, and lung cancer (Tseng 1977, Tseng et al. 1968, Chen et al. 1986).

USEPA has classified arsenic in Group A—Human Carcinogen—and has developed
inhalation (USEPA 1989a) and oral (USEPA 1988) slope factors of
50 $(mg/kg/day)^{-1}$ and 2.0 $(mg/kg/day)^{-1}$, respectively. The inhalation slope
factor is the geometric mean value of slope factors derived from four
occupational exposure studies on two different exposure populations (USEPA
1984). The oral slope factor was based on an epidemiological study in Taiwan
which indicated an increased incidence of skin cancer in individuals exposed
to arsenic in drinking water (Tseng 1977). A risk assessment for
noncarcinogenic effects of arsenic is currently under review by USEPA (1989).
An oral reference dose (RfD) of $1x10^{-3}$ mg/kg/day was calculated for arsenic
based on the same oral epidemiological study (Tseng 1977) which also showed
greater incidence of keratosis and hyperpigmentation in humans (USEPA 1989b).
An uncertainty factor of 1 was used to derive the oral RfD. This RfD is
presently being reconsidered by the RfD workgroup.

Cadmium

Gastrointestinal absorption of cadmium in humans ranges from 5-6% (USEPA
1985a). Pulmonary absorption of cadmium in humans is reported to range from
10% to 50% (CDHS 1986). Cadmium bioaccumulates in humans, particularly in the
kidney and liver (USEPA 1985a,b). Chronic oral or inhalation exposure of

humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans at a renal cortex concentration of cadmium of 200 μg/g (USEPA 1985b). Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lung, kidney, and prostate (USEPA 1985b, Thun et al. 1985). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. When administered by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. Cadmium is a well-documented animal teratogen (USEPA 1985b).

USEPA (1989a,b) classified cadmium as a Group B1 agent (Probable Human Carcinogen) by inhalation. This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies. USEPA (1989a,b) derived an inhalation slope factor of 6.1 $(mg/kg/day)^{-1}$ for cadmium based on epidemiologic studies in which respiratory tract tumors were observed (Thun et al. 1985, USEPA 1985b). Using renal toxicity as an endpoint, and a safety factor of 10, USEPA (1989a,b) has derived two separate oral reference doses (RfD). The RfD associated with oral exposure to drinking water is 5×10^{-4} mg/kg/day, and is based upon the lowest-observed-adverse-effect level (LOAEL) of 0.005 mg/kg in humans (USEPA 1985a, Friberg et al. 1974). The RfD associated with exposure to cadmium in food or other nonaqueous oral exposures is 1×10^{-3} mg/kg/day.

Chromium

Chromium exists in two states, as chromium (III) and as chromium (VI). Following oral exposure, absorption of chromium (III) is low while absorption of chromium (VI) is high (USEPA 1987). Chromium is an essential micronutrient and is not toxic in trace quantities (USEPA 1980). High levels of soluble chromium (VI) and chromium (III) can produce kidney and liver damage following acute oral exposure; target organs affected by chronic oral exposure remain unidentified (USEPA 1984). Chronic inhalation exposure may cause respiratory system damage (USEPA 1984). Further, epidemiological studies of worker populations have clearly established that inhaled chromium (VI) is a human carcinogen; the respiratory passages and the lungs are the target organs (Mancuso 1975, USEPA 1984). Inhalation of chromium (III) or ingestion of chromium (VI) or (III) has not been associated with carcinogenicity in humans or experimental animals (USEPA 1984). Certain chromium salts have been shown to be teratogenic and embryotoxic in mice and hamsters following intravenous or intraperitoneal injection (USEPA 1984).

USEPA has classified inhaled chromium (VI) in Group A--Probable Human Carcinogen by the inhalation route (USEPA 1989). Inhaled chromium (III) and ingested chromium (III) and (VI) have not been classified with respect to carcinogenicity (USEPA 1989). USEPA (1989) developed an inhalation slope factor of 41 $(mg/kg/day)^{-1}$ for chromium (VI) based on an increased incidence of lung cancer in workers exposed to chromium over a 6 year period, and followed for approximately 40 years (Mancuso 1975). USEPA (1989) derived an

oral reference dose (RfD) of $5.0x10^{-3}$ mg/kg/day for chromium (VI) based on a study by MacKenzie et al. (1958) in which no adverse effects were observed in rats exposed to 2.4 mg chromium (VI)/kg/day in drinking water for 1 year. A safety factor of 500 was used to derive the RfD. USEPA (1989) developed an oral RfD of 1 mg/kg/day for chromium (III) based on a study in which rats were exposed to chromic oxide baked in bread; no effects due to chromic oxide treatment were observed at any dose level (Ivankovic and Preussman 1975). A safety factor of 1,000 was used to calculate the oral RfD.

Cyanides

The toxicity of cyanides is strongly dependent on their chemical speciation. Free cyanides are readily absorbed from the gastrointestinal tract, lungs, and skin and, once absorbed, are rapidly distributed throughout the body (USEPA 1985). The toxic effects in humans following acute oral exposure to free cyanides include hyperventilation, vomiting, unconsciousness, convulsions, vascular collapse and cyanosis, and death (USEPA 1985). Inhalation of high concentrations of hydrogen cyanide (HCN) gas results in almost immediate collapse, respiratory arrest, and death within minutes (DiPalma 1971). Airborne hydrogen cyanide concentrations between 99 and 528 mg/m^3 are fatal within 30-60 minutes (NIOSH 1976). There is limited data on chronic exposures of cyanide in humans, although the following effects have been identified in chronic occupationally exposed workers in some epidemiologic studies: neurological dysfunction, lacrimation, abdominal pain, muscular weakness, and shortness of breath (NIOSH 1976). Cyanide appears to be less toxic to animals

A

following chronic exposures than following acute exposures. In animals, chronic oral exposure has produced weight loss, thyroid effects and myelin degeneration (Howard and Hanzal 1955). Cyanide can cause teratogenic effects when subcutaneously administered to hamsters; this teratogenic effect has not been observed in other species although some reproductive toxicity has been noted (USEPA 1985).

USEPA (1989a, 1989b) calculated an oral reference dose (RfD) of 0.02 mg/kg/day for cyanide based on a chronic study by Howard and Hanzal (1955) in which rats were maintained on a diet fumigated with hydrogen cyanide and exhibited weight loss, thyroid effects and myelin degeneration. No observed adverse effects (NOAEL) were noted at the highest dose administered (10.8 mg/kg/day). A NOAEL of 10.8 mg/kg/day and a safety factor of 500 were used to derive the RfD (USEPA 1989a).

Lead

Absorption of lead from the gastrointestinal tract of adult humans is estimated at 8%-45%. In children, absorption from non-paint sources ranges from 30% to 50% (Hammond and Beliles 1980, USEPA 1986). There are other interpretations of the data (Duggan 1983) that suggest this may be as high as 70%. For adult humans, the deposition rate of particulate airborne lead is 30%-50%, and essentially all of the lead deposited is absorbed. Lead is stored in the body in the kidney, liver, and bone (USEPA 1984). The major adverse effects in humans caused by lead include alterations in the

hematopoietic and nervous systems. The toxic effects are generally related to the concentration of this metal in blood. Blood concentration levels of over 80 μg/dl in children and over 100 μg/dl in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possible death. The Centers for Disease Control (CDC 1985) have used the value of 25 μg/dl as an acceptable level of blood lead. Recent information (USEPA 1988), however, indicates that physiological and/or biochemical effects can occur even at lower levels. These include enzyme inhibition (16 μg/dl), elevated erythrocyte protoporphyrin (15 μg/dl), interference with Vitamin D metabolism, cognitive dysfunction in infants (10 to 15 μg/dl), electrophysiological dysfunction (6 μg/dl), and reduced childhood growth (4 μg/dl). Decreased fertility, fetotoxic effects, and skeletal malformations have been observed in experimental animals exposed to lead (USEPA 1984). Chronic oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) has been associated in experimental animals with increased renal tumors. Doses of lead that induced kidney tumors were high and were beyond the lethal dose in humans (USEPA 1985).

USEPA classified certain lead salts in Group B2—Probable Human Carcinogen, although no slope factor has been established (USEPA 1989a). This category applies to those agents for which there is sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in humans. USEPA (1988) has recently proposed a maximum contaminant level goal (MCLG) of zero for lead. USEPA (1989a) has considered it inappropriate to develop a reference dose (RfD) for inorganic lead and lead compounds, since

many of the health effects associated with lead intake occur essentially
without a threshold. The new proposed MCLG is based on subtle effects of lead
at low blood levels, the overall Agency goal of reducing lead exposures, and
the probable carcinogenicity of lead at very high doses. Underlying this
proposal was the assumption that blood lead levels in the range of 10-15 μg/dl
are associated with serious effects. Additionally USEPA noted that existing
body burdens of lead were already in the range where adverse effects could
result.

An alternative approach is also undergoing review by USEPA to evaluate
potential subchronic lead exposures to young children. This approach is based
on a linear pharmacokinetic model used by USEPA's Office of Air Quality
Planning and Standards (OAQPS) for lead air quality standard setting (USEPA
1989b). The model, based on work by Harley and Kneip (1985), takes into
account the uptake, retention and excretion of lead. It is referred to as the
"Integrated Uptake/Biokinetic Model", and it estimates blood lead levels.

Mercury

In humans, inorganic mercury is absorbed following inhalation and oral
exposure, however only 7% to 15% of administered inorganic mercury is absorbed
following oral exposure (USEPA 1984, Rahola et al. 1971, Task Group on Metal
Accumulation 1973). Organic mercury is almost completely absorbed from the
gastrointestinal tract and is assumed to be well absorbed via inhalation in
humans (USEPA 1984). A primary target organ for inorganic compounds is the

kidney. Acute and chronic exposures of humans to inorganic mercury compounds have been associated with anuria, polyuria, proteinuria, and renal lesions (Hammond and Beliles 1980). Chronic occupational exposure of workers to elemental mercury vapors (0.1 to 0.2 mg/m^3) has been associated with mental disturbances, tremors, and gingivitis (USEPA 1984). Animals exposed to inorganic mercury for 12 weeks have exhibited proteinuria, nephrotic syndrome and renal disease (Druet et al. 1978). Rats chronically administered inorganic mercury (as mercuric acetate) in their diet have exhibited decreased body weights and significantly increased kidney weights (Fitzhugh et al. 1950). The central nervous system is a major target for organic mercury compounds. Adverse effects in humans, resulting from subchronic and chronic oral exposures to organic mercury compounds, have included destruction of cortical cerebral neurons, damage to Purkinje cells, and lesions of the cerebellum. Clinical symptoms following exposure to organic mercury compounds have included paresthesia, loss of sensation in extremities, ataxia, and hearing and visual impairment (WHO 1976). Embryotoxic and teratogenic effects, including malformations of the skeletal and genitourinary systems, have been observed in animals exposed orally to organic mercury (USEPA 1984). Both organic and inorganic compounds are reported to be genotoxic in eukaryotic systems (Leonard et al. 1984).

USEPA (1989) has reported an oral RfD for alkyl and inorganic mercury of 3×10^{-4} mg/kg/day based on studies investigating central nervous system effects in humans exposed to mercury (USEPA 1980, 1984); an uncertainty factor of 10 was used to develop the RfD. USEPA (1989) has also reported an oral reference

dose of $3x10^{-4}$ mg/kg/day for inorganic mercury based on a chronic rat study in which kidney effects were observed (Fitzhugh et al. 1950). An uncertainty factor of 1,000 was used to derive the RfD.

Nickel

Nickel compounds can be absorbed following inhalation, ingestion, or dermal exposure. The amount absorbed depends on the dose administered and the chemical and physical form of the particular nickel compound (USEPA 1986). Dermal exposure of humans to nickel produces allergic contact dermatitis (USEPA 1986). Adverse effects associated with acute exposure in animals have included depressed weight gain, altered hematological parameters, and increased iron deposition in blood, heart, liver, and testes (USEPA 1987). Chronic or subchronic exposure of experimental animals to nickel has been associated with reduced weight gain, degenerative lesions of the male reproductive tract, asthma, nasal septal perforations, rhinitis, sinusitis, hyperglycemia, decreased prolactin levels, decreased iodine uptake, and vasoconstriction of the coronary vessels (USEPA 1986). Teratogenic and fetotoxic effects have been observed in the offspring of exposed animals (USEPA 1986). Inhalation exposure of experimental animals to nickel carbonyl or nickel subsulfide induces pulmonary tumors (USEPA 1986). Several nickel salts cause localized tumors when administered by subcutaneous injection or implantation. Epidemiological evidence indicates that inhalation of nickel refinery dust and nickel subsulfide is associated with cancers of the nasal cavity, lung, larynx, kidney, and prostate (USEPA 1986).

Nickel refinery dust and nickel subsulfide are both categorized in Group A--Human Carcinogens (USEPA 1989). These classifications are based on an increased incidence of lung and nasal tumors observed in workers occupationally exposed to nickel refinery dust (USEPA 1986). These materials have inhalation slope factors of 0.84 $(mg/kg/day)^{-1}$ and 1.7 $(mg/kg/day)^{-1}$, respectively (USEPA 1989). Nickel carbonyl is categorized in Group B2--Probable Human Carcinogen; however, a slope factor has not been derived for nickel carbonyl (USEPA 1989). USEPA (1989) derived an oral reference dose (RfD) for nickel of 2×10^{-2} mg/kg/day based on a study by Ambrose et al. (1976) in which rats administered 5 mg/kg/day (NOAEL) nickel in the diet for 2 years did not experience decreased weight gain, which was observed in animals administered 50 mg/kg/day (LOAEL). A safety factor of 300 was used to calculate the RfD.

Selenium

Results of studies with humans and experimental animals indicate that certain selenium compounds are readily absorbed from the gastrointestinal tract following oral exposure (USEPA 1984). The pulmonary absorption of selenium following inhalation exposure has not been well studied, although there are reports suggesting that selenium is absorbed to some extent by this route (USEPA 1984). Selenium is an essential element and therefore is nontoxic at doses necessary for normal health and nutrition. NAS (1980) reported that an adequate and safe selenium intake for an adult human ranges from 0.05 mg/day

to 0.2 mg/day. However, exposure to selenium at levels that exceed these levels has been associated with adverse health effects. Such effects observed in experimental animals following subchronic or chronic oral exposure to various selenium compounds have included anemia, reduced growth, increased mortality, and lesions of the liver, heart, kidney, and spleen (USEPA 1984). In humans, chronic oral exposure to selenium has been associated with alopecia, dermatitis, discoloration of the skin, loss of fingernails, muscular dysfunction, convulsions, paralysis, and increased incidences of dental caries (USEPA 1984, Yang et al. 1983). Headaches and respiratory irritation have been noted in humans following acute inhalation exposure (USEPA 1984); dermatitis and gastrointestinal disturbances have resulted from occupational exposure (Glover 1967). Studies with a variety of animals have suggested that selenium may be teratogenic; however, these studies are limited in that exposure levels are not well characterized (USEPA 1984).

Oral and inhalation reference doses (RfD) of 3.0×10^{-3} mg/kg/day and 1.0×10^{-3} mg/kg/day, respectively, have been derived by USEPA (1984, 1989). The oral RfD value was based on a study by Yang et al. (1983) in which humans exposed to selenium in the diet at doses of 3.2 mg/day developed loss of hair, loss of fingernails, dermatitis, and muscular dysfunction. By applying an uncertainty factor of 15 to the LOAEL of 3.2 mg/day, USEPA (1989) determined the oral RfD value of 3×10^{-3} mg/kg/day. The oral RfD is currently under review by the oral RfD Work Group at USEPA (1989). The inhalation RfD value was based on an occupational study by Glover (1967) in which workers exposed to airborne concentrations of selenium developed dermatitis and

gastrointestinal disturbances. An uncertainty factor of 10 was used to determine the inhalation RfD (USEPA 1989).

Silver

Silver in various forms is absorbed to a limited extent following oral and inhalation exposures (USEPA 1985). The acute toxic effects in humans following oral exposure to silver include corrosive damage to the GI tract leading to shock, convulsions, and death. In animals, acute exposure has been shown to affect the central nervous system and to cause respiratory paralysis (Hill and Pillsbury 1939). The primary effect of silver in humans following chronic exposures is argyria, a permanent bluish-metallic discoloration of the skin and mucous membranes, which can be either localized or generalized. Silver also accumulates in the blood vessels and connective tissue (USEPA 1985).

USEPA (1989) derived an oral reference dose (RfD) of $3.0x10^{-3}$ mg/kg/day for silver based on the human case reports of Gaul and Staud (1935), Blumberg and Carey (1934), and East et al. (1980). In these studies, argyria was observed at an average dose of silver of 0.0052 mg/kg/day, to which an uncertainty factor of 2 was applied.

Zinc

Zinc is absorbed in humans following oral exposure; however, insufficient data are available to evaluate absorption following inhalation exposure (USEPA 1984). Zinc is an essential trace element that is necessary for normal health and metabolism and therefore is nontoxic in trace quantities (Hammond and Beliles 1980). Exposure to zinc at concentrations that exceed recommended levels, however, has been associated with a variety of adverse effects. Chronic and subchronic inhalation exposure of humans to zinc has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by fever, chills, coughing, dyspnea, and muscle pain (USEPA 1984). Chronic oral exposure of humans to zinc may cause anemia and altered hematological parameters (Pories et al. 1967, Prasad et al. 1975). Reduced body weights have been observed in studies in which rats were administered zinc in the diet. There is no evidence that zinc is teratogenic or carcinogenic (USEPA 1984).

USEPA (1989) has derived an oral reference dose (RfD) of 2×10^{-1} mg/kg/day based on studies in which anemia and reduced blood copper were observed in humans exposed to oral zinc doses of 2.14 mg/kg/day (Pories et al. 1967, Prasad et al. 1975). A safety factor of 10 was used in developing the RfD.

5.0 HEALTH RISK CHARACTERIZATION

In this section, chemical concentrations found in environmental media at or near the site and at receptor locations are compared with applicable or relevant and appropriate requirements (ARARs) or other guidance that have been developed for the protection of human health or the environment. In addition, quantitative risk estimates are also calculated to evaluate the potential for adverse effects on human health.

5.1 HEALTH-BASED APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS)

In this section, ARARs or other guidance are first identified for the chemicals of concern. Where chemical-specific or ambient ARARs are available for an environmental medium, they are compared with average and maximum concentrations observed in that medium at points of potential exposure. USEPA interim guidance on ARARs (USEPA 1987c) defines them as follows:

"Applicable requirements" means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that specifically address a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site. "Applicability" implies that the remedial action or the circumstances at the site satisfy all of the jurisdictional prerequisites of a requirement.

"Relevant and appropriate requirements" means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that, while not "applicable" to a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site, address problems or situations sufficiently similar to those encountered at the CERCLA site that their use is well suited to the particular site.

The relevance and appropriateness of a requirement can be judged by comparing a number of factors, including the characteristics of the remedial action, the hazardous substances in question, or the physical circumstances of the site, with those addressed in the requirement. It is also helpful to look at the objective and origin of the requirement.

A requirement that is judged to be relevant and appropriate must be complied with to the same degree as if it were applicable. However, there is more discretion in this determination: it is possible for only _part_ of a requirement to be considered relevant and appropriate, the rest being dismissed if judged not to be relevant and appropriate in a given case.

Non-promulgated advisories or guidance documents issued by federal or state governments do not have the status of potential ARARs. However, they may be considered and may be used in setting the necessary level of cleanup for protection of health or the environment.

Only those ARARs or advisories or guidance that are ambient or chemical-specific requirements [i.e., those requirements which "set health or risk-based concentration limits or ranges in various environmental media for specific hazardous substances, pollutants, or contaminants" (USEPA 1987c)], as opposed to ARARs which are classified as action-specific or locational requirements, are used in this risk assessment.

The classes of ambient or chemical-specific health-based ARARs or guidance that are considered pertinent to this assessment for the SCP site are discussed below by environmental medium and summarized in Table 5-1.

5.1.1 Ground Water

Federal ARARs for drinking water are Maximum Contaminant Levels (MCLs) under the Safe Drinking Water Act (SDWA), and human health-based Ambient Water Quality Criteria (AWQC) set under the Clean Water Act. MCLs are enforceable drinking water standards and are not strictly health-based. Technological and economic feasibility are also taken into account in developing MCLs. According to USEPA guidance on the use of MCLs as ARARs (USEPA 1987c), MCLs are relevant and appropriate requirements against which to evaluate groundwater quality.

Maximum Contaminant Level Goals (MCLGs) are promulgated under the SDWA as chemical-specific health criteria used in setting MCLs and other enforceable drinking water standards. An MCLG for a toxic chemical is based only on

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TABLE 5-1

POTENTIAL FEDERAL AND STATE HEALTH-BASED ARARs AT THE SCP SITE
(Concentrations in ug/l, unless otherwise noted)

Compound	Federal Water — ARAR: Safe Drinking Water Act Maximum Contaminant Level (MCL)	Federal Water — Other Guidance: Maximum Contaminant Level Goal (MCLG)	Federal Water — Other Guidance: Ambient Water Quality Criteria for Protection of Human Health (a)	Federal Soil — Other Guidance: TSCA PCB Spill Policy (mg/kg)	State Water (ARARs) — Maximum Contaminant Level	State Water (ARARs) — Surface Water Quality Criteria	State Water (ARARs) — Ground Water Quality Standards	State Soil — Other Guidance: NJDEP Soil Cleanup Objectives (mg/kg)
VOLATILE ORGANIC CHEMICALS								
Benzene	5	0	0 (0.67)	--	1	--	1	1 (o)
Chlorobenzene	100 (s)	100 (s)	488	--	4	--	4	1 (o)
Chloroform	100 (b)	--	0 (0.19)	--	100 (t,b)	--	100 (b)	1 (o)
1,1-Dichloroethane	--	--	--	--	--	--	--	1 (o)
1,2-Dichloroethane	5	0	0 (0.94)	--	2	--	2	1 (o)
1,1-Dichloroethylene	7	7	0 (0.033)	--	2	--	2	1 (o)
1,2-trans-Dichloroethylene	100 (s)	70 (s)	--	--	10	--	10	1 (o)
Ethylbenzene	700 (s)	680	2,400	--	--	--	--	1 (o)
Methylene chloride	--	--	--	--	2	--	2	1 (o)
Methyl ethyl ketone	--	--	--	--	--	--	--	1 (o)
1,1,2,2-Tetrachloroethane	--	--	0 (0.17)	--	--	--	--	1 (o)
Tetrachloroethylene	5 (s)	0 (s)	0 (0.88)	--	1		1	1 (o)
Toluene	2,000 (s)	2,000 (s)	15,000	--	--	--	--	1 (o)
1,1,1-Trichloroethane	200	200	19,000	--	26	--	26	1 (o)
Trichloroethene	5	0	0 (2.8)	--	1	--	1	1 (o)
Vinyl chloride	2	0	0 (2)	--	2	--	2	1 (o)
Xylenes	10,000 (s)	10,000 (d)	--	--	44 (d)	--	44 (d)	1 (o)
PESTICIDES								
Aldrin	--	--	0 (0.0012)	--	--	0.0019	0.003 (g)	--
Dieldrin	--	--	0 (0.0011)	--	--	0.0019	0.003 (g)	--
Polychlorinated biphenyls (PCBs)	0.5 (s)	0 (s)	0 (<0.00126)	25 (q), 10 (r)	0.5 (s)	0.014 (e), 0.030 (f)	0.001 (g)	1 - 5
BASE NEUTRALS AND ACIDS								
Bis(2-ethylhexyl)phthalate	--	--	21,000	--	--	--	--	10 (p)
Butylbenzylphthalate	--	--	--	--	--	--	--	10 (p)
1,2-Dichlorobenzene	--	620	470	--	--	--	--	10 (p)
Di-n-butyl phthalate	--	--	19,800	--	--	--	--	1 (o)
Di-n-octyl phthalate	--	--	44,000	--	--	--	--	10 (p)
Nitrobenzene	--	--	--	--	--	--	--	10 (p)
PAHs (Total)	--	--	3,100	--	--	--	--	10 (p)
Phenol	--	--	3,500	--	--	--	3,500 (g), 300 (h)	10 (p)

TABLE 5-1 (Continued)

POTENTIAL FEDERAL AND STATE HEALTH-BASED ARARs AT THE SCP SITE
(Concentrations in ug/l, unless otherwise noted)

Compound	Federal Water: ARAR: Safe Drinking Water Act Maximum Contaminant Level (MCL)	Federal Water: Other Guidance: Maximum Contaminant Level Goal (MCLG)	Federal Water: Other Guidance: Ambient Water Quality Criteria for Protection of Human Health (a)	Federal Soil: Other Guidance: Secondary Drinking Water Standards	State Water (ARARs): NJ Safe Drinking Water Act Maximum Contaminant Level	State Water (ARARs): Surface Water Quality Criteria	State Water (ARARs): Ground Water Quality Standards	State Soil: Other Guidance: NJDEP Soil Cleanup Objectives (mg/kg)
INORGANICS								
Antimony	--	--	146	--	--	--	--	--
Arsenic	50	50 (s)	0 (0.025)	--	-- (t)	50 (e)	50 (g)	20
Beryllium	--	--	0 (0.0039)	--	--	--	--	--
Cadmium	10	5 (s)	10	--	-- (t)	10 (e)	10 (g)	3
Chromium	50 (100 - s)	50 (100 - s)	179,000; 50 (l,m)	--	-- (t)	50 (e)	50 (g, i)	100
Copper	(300 - s)	1,300 (s)	1,000 (c)	--	--	--	1,000 (h)	170
Cyanide	--	--	200	--	--	--	200 (g)	12
Lead	50	20 (0 -s)	50	--	-- (t)	50 (e)	50 (g)	250-1,000
Mercury	2	2 (s)	10	--	-- (t)	2	2 (g)	1
Nickel	--	--	15.4	--	--	--	--	100
Selenium	10	50 (s)	10	--	-- (t)	10 (e)	10 (g)	4
Silver	50	--	50	--	-- (t)	50	50 (g)	5
Zinc	--	--	5,000 (c)	--	--	--	--	350

(a) The criterion value, which is zero for all potential carcinogens, is listed for all chemicals in the table. The concentration value given in parentheses for potential carcinogens corresponds to a risk of 10-6, which is the midpoint of the concentration corresponding to risks of 10-5 and 10-7 given in water quality criteria documents. To obtain concentrations corresponsing to risks of 10-7, the 10-6 concentrations should be divided by 10. The criteria include contributions of exposure from ingestion of drinking water only.
(b) This MCL applies to total trihalomethenes.
(c) Criteria designated as organoleptic are based on taste and odor effects, not human health effects. Health-based water quality criteria are not available for these chemicals.
(d) Applies to total xylenes.
(e) Freshwater (FW2 classification).
(f) Saltwater (SE classification).
(g) Primary standards for groundwater classification GW2.
(h) Secondary standards for groundwater classification GW2.
(i) Chromium VI and compounds.
(l) Applied to inorganic arsenic (III) compounds.
(m) Values for chromium (III) and chromium (VI), respectively.
(n) Applies to free cyanide.
(o) Applies to total volatile organics.
(p) Applies to total base neutrals and acids.
(q) Applies to restricted access areas.
(r) Applies to non-restricted access areas.
(s) Proposed.
(t) In the absence of a New Jersey State promulgated MCL for a specified compound, NJDEP adopts the federal MCL.

Sources: Federal: Safe Drinking Water Act MCL (40 CFR Parts 141 and 142; Fed. Reg. 52:25690-25694)
Ambient Water Quality Criteria (Section 304(a) Clean Water Act; criteria for protection of human health from ingestion calculated in USEPA 1986.
Superfund Public Health Evaluation Manual, EPA 540/1-86/060.
TSCA PCB Spill Policy (40 CFR 761)
State: Maximum Contaminant Level (NJAC 7:10-16)
Surface Water Quality Standards (NJAC 7:9-4)
Ground Water Quality Standards (NJAC 7:9-6)
NJDEP Soil Cleanup Objectives (NJDEP, Summary of Approached to Soil Cleanup Levels, February 19, 1987)

health considerations (i.e., technological and economic considerations are not included) and represents a level at which no adverse effects occur (USEPA 1987c).

State drinking water and ground water standards that may be health-based ARARs for specific chemicals at the SCP site include Ground Water Quality Criteria established under the New Jersey Administrative Code (NJAC), Section 7:9-6; and New Jersey Safe Drinking Water Act Maximum Contaminant Levels (NJAC 7:10-16). The New Jersey Ground Water Quality Criteria establish allowable levels of certain chemical pollutants for ground water in New Jersey aquifers. The New Jersey Department of Environmental Protection (NJDEP) has also developed recommended MCLs for organic and inorganic chemicals under the Safe Drinking Water Act. These MCLs were adopted on January 3, 1989 as NJAC 7:10-16.

5.1.2 Soil and Sediment

With the exception of PCBs, USEPA has not established or proposed any standards or criteria for acceptable chemical concentrations in soil or sediment. The limits for PCBs developed under the TSCA PCB Spill Policy (40 CFR Part 761) established requirements for the cleanup of PCB spills of materials containing 50 ppm PCBs or greater. The TSCA policy requires more stringent clean-up for spills involving higher concentrations of PCBs located in areas where their release would pose the greatest potential for significant human exposure. Contaminated soil in restricted access areas should be cleaned to 25 ppm PCBs by weight; soil in non-restricted access areas should

be cleaned to 10 ppm provided that the soil is excavated to a minimum depth of ten inches and is replaced with clean soil (soil containing less than 1 ppm PCBs). Restricted access areas must be more than 0.1 kilometer away from a residential/commercial area; otherwise they are considered to be a residential/commercial area. The SCP site might be considered a restricted access area, depending on future land use in the vicinity of the property and on the site itself. More and less stringent clean-up measures are allowable on a site-specific basis.

USEPA (1989b) has also developed an interim soil cleanup level for total lead in soil of 500-1,000 mg/kg based on a recommendation from the Centers for Disease Control (CDC 1985). USEPA currently considers this guidance range protective for direct soil contact in residential land use settings, but notes that efforts are underway to develop further guidance related to remedial actions involving lead at Superfund sites (USEPA 1989b).

The State of New Jersey Department of Environmental Protection has also established soil cleanup objectives under the Environmental Cleanup Responsibility Act (ECRA). Cleanup levels, or ranges, have been defined for 14 metals, PCBs, total volatiles, total base-neutral extractable organics, and petroleum hydrocarbons.

5.1.3 Surface Water

As mentioned above in Section 8.1.1, federal ARARs for surface water used as drinking water are MCLs under the Safe Drinking Water Act, and human health-based AWQC under the Clean Water Act. The state MCLs would also be ARARs, if the surface waters near the site (Peach Island Creek) were a potential source of drinking water. However, because of the salinity of the water (4.2 parts per thousand), it is very unlikely that these surface waters would ever be used as a source of drinking water. Therefore, these ARARs and potential ARARs are not relevant for surface water near the SCP site. State Surface Water Criteria (NJAC 7:9-6) are, however, applicable for the surface water at the site. These values (maximum levels are set for 18 toxic pollutants and classes of pollutants) are set both to protect human health and environmental resources.

5.1.4 Air

Under the Clean Air Act, USEPA has promulgated national ambient air quality standards (NAAQS) for seven criteria pollutants: nitrogen dioxide, sulfur dioxide, carbon monoxide, "respirable" particulate matter (PM_{10}), total nonmethane hydrocarbons, ozone, and lead. The State of New Jersey also has ambient standards for six of these criteria pollutants (no standards exist for nonmethane hydrocarbons), but not for any of the toxic contaminants of concern at the SCP site.

5.1.5 Violations of ARARs or Other Guidance

5.1.5.1 Soil

The soil sampling data from the site indicate that numerous chemicals exceed the NJDEP ECRA soil cleanup objectives. Both the maximum and geometric mean concentrations of total volatile organics, and total base neutral and acid extractable compounds, detected in shallow and medium depth soil exceeded the ECRA limits (1 mg/kg and 10 mg/kg, respectively). In deep soil, the maximum detected volatile organic and base neutral/acid levels exceeded the ECRA objectives. In very deep soil, both the maximum and average volatile organic levels exceeded the ECRA objective, while the maximum levels of base neutral/acid extractable compounds exceeded the ECRA objective. The primary volatiles that accounted for the soils at the site exceeding the ECRA total volatiles level (1 mg/kg) were chlorobenzene, 1,1-dichloroethane, ethylbenzene, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene, and xylenes. The base neutrals and acid extractables that predominantly accounted for the soils exceeding the ECRA objectives were PAHs, butyl benzyl phthalate, 1,2-dichlorobenzene, and phenol.

The ECRA objective was also exceeded by PCBs at all soil depths sampled, for both the maximum and geometric mean concentrations. (The maximum PCB levels in shallow and medium depth soils also exceeded the TSCA PCB spill clean-up level for a restricted access area.) Among the inorganics detected in shallow and medium soil, the average and maximum levels of cadmium, copper, lead,

mercury, and zinc exceeded their ECRA objectives. The maximum concentrations of these inorganics in deep soil also exceeded the ECRA levels. The maximum detected concentrations of arsenic, chromium and silver in shallow and medium soil exceeded the ECRA levels, while the maximum detected selenium level in shallow soil and of nickel in medium soil exceeded their ECRA levels. The ECRA objectives were not exceeded by any inorganics in very deep soil.

The maximum lead concentrations detected in shallow and medium depth soil exceeded the USEPA (1989b) guidance 500-1,000 mg/kg interim soil cleanup range for residential land use. The geometric mean concentrations at these depths, as well as in deep and very deep soil, were below the cleanup range, while the maximum detected concentration in deep soil was within this range. The maximum regional background soil concentration for lead shown in Table 2-1 falls within the 500-1,000 mg/kg range.

5.1.5.2 Ground Water

The concentrations of numerous VOCs, PCBs, and several inorganics in ground water exceed ARARs and other guidance. Ground water ARARs include federal and state maximum contaminant levels (MCLs), and state ground water quality standards (GWQS). Other guidance includes federal ambient water quality criteria (AWQC) for the protection of human health and federal maximum contaminant level goals (MCLGs). Tables 5-2, 5-3, and 5-4 compare ARARs and other guidance to water quality in the water table, till, and bedrock aquifers, respectively. Values which exceed ARARs and other guidance are

TABLE 5-2

EXCEEDANCES OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs) AND OTHER GUIDANCE FOR GROUND WATER: WATER TABLE AQUIFER

Chemical	Federal ARAR: MCL Geometric Mean	Maximum	Federal Other Guidance: MCLG Geometric Mean	Maximum	Federal Other Guidance: AWQC Geometric Mean	Maximum	State ARAR: MCL Geometric Mean	Maximum	State ARAR: SWQC Geometric Mean	Maximum	State ARAR: GWQS Geometric Mean	Maximum
VOCs:												
Benzene	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
Chlorobenzene	--	P *	--	P *	--	X	X	X *	NA	NA	X	X *
Chloroethane	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chloroform	--	X *	NA	NA	X *	X *	--	X *	NA	NA	--	X *
1,1-Dichloroethane	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2-Dichloroethane	X	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
1,1-Dichloroethylene	--	X	--	X	--	X *	--	X *	NA	NA	--	X *
1,2-trans-Dichloroethylene	P *	P *	P *	P *	NA	NA	X *	X *	NA	NA	X *	X *
Ethylbenzene	--	P	--	X	--	X	NA	NA	NA	NA	NA	NA
Methylene chloride	NA	NA	NA	NA	NA	NA	X *	X *	NA	NA	X *	X *
Methyl ethyl ketone	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,1,2,2-Tetrachloroethane	NA	NA	NA	NA	X *	X *	NA	NA	NA	NA	NA	NA
Tetrachloroethylene	P	P *	P *	P *	X *	X *	X *	X *	NA	NA	X *	X *
Toluene	P	P *	P	P *	--	X	NA	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	--	X *	--	X *	--	X	X	X *	NA	NA	X *	X *
Trichloroethylene	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
Vinyl chloride	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
Xylenes	--	P	--	P	NA	NA	X	X *	NA	NA	X	X *
Pesticides/PCBs:												
PCBs	P	P *	P	P *	X *	X *	P	P *	X *	X *	X *	X *
Semi-Volatiles:												
Total PAHs	NA	NA	NA	NA	--	--	NA	NA	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate	NA	NA	NA	NA	--	--	NA	NA	NA	NA	NA	NA
Butylbenzyl phthalate	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2-Dichlorobenzene	NA	NA	--	--	--	--	NA	NA	NA	NA	NA	NA
Nitrobenzene	NA	NA	--	NA	--	X	NA	NA	NA	NA	NA	NA
Di-n-butyl phthalate	NA	NA	NA	NA	--	--	NA	NA	NA	NA	NA	NA
Phenol	NA	NA	NA	NA	--	X	NA	NA	NA	NA	X	X
Inorganics:												
Arsenic	--	X *	--	P *	X *	X *	--	X *	--	X *	--	X *
Beryllium	NA	NA	NA	NA	X *	X *	NA	NA	NA	NA	NA	NA
Cadmium	--	X	--	P	--	X	--	X	--	X	--	X
Chromium	--	X,P	--	X,P	--	--	--	X	--	X	--	X
Copper	P	P	--	P	--	X	NA	NA	NA	NA	--	X
Cyanide	NA	NA	NA	NA	--	--	NA	NA	NA	NA	--	--
Lead	--	X *	--	X,P *	--	X *	--	X *	--	X *	--	X *
Mercury	--	X	--	P	--	--	--	X	--	X	--	X
Nickel	NA	NA	NA	NA	X	X *	NA	NA	NA	NA	NA	NA
Zinc	NA	NA	NA	NA	--	--	NA	NA	NA	NA	NA	NA

X = Exceeds promulgated standard or other guidance.
P = Exceeds proposed standard or guidance. Proposed standards are not ARARs.
* = Exceeds standard by greater than a factor of 10.
NA = No applicable value.
-- = Concentration does not exceed ARAR or other guidance.

Abbreviations:
  MCL = Maximum Contaminant Level
  MCLG = Maximum Contaminant Level Goal
  AWQC = Ambient Water Quality Criteria

TABLE 5-3

EXCEEDANCES OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs) AND OTHER GUIDANCE
FOR GROUND WATER: TILL AQUIFER

| | Federal | | | | | | State | | | | | |
| | ARAR: MCL | | Other Guidance: MCLG | | Other Guidance: AWQC | | ARAR: MCL | | ARAR: SWQC | | ARAR: GWQS | |
Chemical	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum
VOCs:												
Chlorobenzene	--	--	--	--	--	--	X	X *	NA	NA	X	X
Chloroform	X	X *	NA	NA	X *	X *	X	X *	NA	NA	X	X *
1,1-Dichloroethane	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1,2-Dichloroethane	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
1,1-Dichloroethylene	X	X *	X	X *	X *	X *	X	X *	NA	NA	X	X *
1,2-trans-Dichloroethylene	--	P	--	P	NA	NA	X	X *	NA	NA	X	X *
Methylene chloride	NA	NA	NA	NA	NA	NA	X *	X *	NA	NA	X *	X *
Tetrachloroethylene	P	P *	P *	P *	X *	X *	X *	X *	NA	NA	X *	X *
Toluene	--	--	--	--	--	--	NA	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	--	X	--	X	--	--	X	X *	NA	NA	X	X *
Trichloroethylene	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
Vinyl chloride	--	X *	--	X *	--	X *	--	X *	NA	NA	--	X *
Pesticides/PCBs:												
PCBs	--	P	--	P *	--	P *	--	P	--	X *	--	X *
Semi-Volatiles:												
1,2-Dichlorobenzene	NA	NA	--	--	--	--	NA	NA	NA	NA	NA	NA
Nitrobenzene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Phenol	NA	NA	NA	NA	--	--	NA	NA	NA	NA	--	--
Inorganics:												
Copper	--	--	--	--	--	--	NA	NA	NA	NA	--	--
Zinc	NA	NA	NA	NA	--	--	NA	NA	NA	NA	NA	NA

X = Exceeds promulgated standard or other proposed guidance.
P = Exceeds proposed standard or guidance. Proposed standards are not ARARs.
* = Exceeds standard by greater than a factor of 10.
NA = No applicable value.
-- = Concentration does not exceed ARAR or other guidance.

Abbreviations:
  MCL = Maximum Contaminant Level
  MCLG = Maximum Contaminant Level Goal
  AWQC = Ambient Water Quality Criteria
  SWQC = Surface Water Quality Criteria
  GWQC = Ground Water Quality Standard

TABLE 5-4

EXCEEDANCES OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs) AND OTHER GUIDANCE
FOR GROUND WATER: BEDROCK AQUIFER

| | Federal | | | | | | State | | | | | |
| Chemical | ARAR: MCL | | Other Guidance: MCLG | | Other Guidance: AWQC | | ARAR: MCL | | ARAR: SWQC | | ARAR: GWQS | |
	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum	Geometric Mean	Maximum
VOCs:												
Chloroform	X	X	NA	NA	X *	X *	X	X	NA	NA	X	X
1,2-Dichloroethane	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
1,1-Dichloroethylene	--	--	--	--	--	X *	--	X	NA	NA	--	X
1,2-trans-Dichloroethylene	--	--	--	--	NA	NA	--	--	NA	NA	--	--
Methylene chloride	NA	NA	NA	NA	NA	NA	--	X *	NA	NA	--	X *
Tetrachloroethylene	--	--	--	P	--	X	--	X	NA	NA	--	X
Toluene	--	--	--	--	--	--	NA	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	--	--	--	--	--	--	--	--	NA	NA	--	--
Trichloroethylene	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
Vinyl chloride	X *	X *	X *	X *	X *	X *	X *	X *	NA	NA	X *	X *
Inorganics:												
Chromium	--	--	--	--	--	--	--	--	--	--	--	--
Copper	--	--	--	--	--	--	NA	NA	NA	NA	--	--
Lead	--	--	--	P	--	--	--	--	--	--	--	--
Zinc	NA	NA	NA	NA	--	--	NA	NA	NA	NA	NA	NA

X = Exceeds promulgated standard or other guidance.
P = Exceeds proposed standard or guidance. Proposed standards are not ARARs.
* = Exceeds standard by greater than a factor of 10.
NA = No applicable value.
-- = Concentration does not exceed ARAR or other guidance.

Abbreviations:
  MCL = Maximum Contaminant Level
  MCLG = Maximum Contaminant Level Goal
  AWQC = Ambient Water Quality Criteria
  SWQC = Surface Water Quality Criteria
  GWQC = Ground Water Quality Standard

noted, as are those cases in which the values exceed the requirements by more than a factor of ten (noted with a "*").

Both the geometric mean and maximum concentrations of benzene in the water table aquifer exceeded its federal and state MCL, the state GWQS, the federal AWQC for protection of human health and the MCLG. The maximum chlorobenzene concentration in the water table aquifer exceeded the AWQC and the MCLG while both the mean and maximum concentrations in the water table and till aquifers exceeded the state MCL and state GWQS. The mean and maximum 1,2-dichloroethane and trichloroethylene concentrations in the water table, till, and bedrock aquifers exceeded their federal and state MCLs, their state GWQS, their MCLGs, and their AWQC. The mean and maximum chloroform concentrations in the water table and till aquifers exceeded the AWQC, while the maximum water table chloroform level also exceeded the federal and state MCL and state GWQS. The mean and maximum chloroform concentrations in the till and bedrock aquifers exceeded both the federal and state MCL, the state GWQS, and the federal AWQC. The mean and maximum 1,1,2,2-tetrachloroethane concentrations in the water table aquifer exceeded the AWQC. The maximum 1,1,1-trichloroethane concentrations in the water table and till aquifers exceeded the federal MCL and MCLG, while the maximum water table level also exceeded the AWQC. Both the mean and maximum 1,1,1-trichloroethane concentrations in the water table and till aquifers exceeded the state MCL and GWQS. The state MCL and GWQS were also exceeded by the mean and maximum 1,2-trans-dichloroethylene levels in both the water table and till aquifers. The mean and maximum 1,2-trans-dichloroethylene concentrations in the water table

aquifer exceeded the proposed federal MCL and proposed MCLG. The maximum 1,2-trans-dichloroethylene concentration in the till aquifer exceeded the proposed federal MCL and the proposed MCLG. The maximum ethylbenzene and toluene levels in the water table exceeded their AWQC. The average and maximum toluene levels in the water table exceeded the MCLG and the proposed federal MCL. The maximum ethylbenzene concentration in the water table also exceeded the proposed federal MCL and MLG. The mean and maximum xylene levels in the water table exceeded the state MCL and GWQS while the maximum xylene level in the water table exceeded the MCLG and proposed federal MCL. The mean and maximum tetrachloroethylene levels in the water table and till aquifers exceeded the state MCL, the proposed federal MCL, the state GWQS, the proposed MCLG, and the AWQC. The maximum tetrachloroethylene concentration in the bedrock aquifer exceeded the proposed MCLG, the AWQC, and the state MCL and GWQS. The mean and maximum methylene chloride levels in the water table and till aquifers, along with the maximum concentration in the bedrock aquifer, exceeded the state MCL and GWQS. The maximum 1,1-dichloroethylene level in the water table aquifer, and the mean and maximum levels in the till aquifer exceeded the state and federal MCL, the state GWQS, the MCLG, and the federal AWQC. The maximum concentration of 1,1-dichloroethylene in the bedrock aquifer exceeded the state MCL and GWQS and the AWQC. The mean and maximum vinyl chloride levels in the water table and bedrock aquifers and the maximum vinyl chloride level in the till aquifer exceeded the federal and state MCL, the state GWQS, the MCLG, and the federal AWQC.

Among the pesticides/PCBs and semi-volatiles, ARARs and other guidance were exceeded for several chemicals. The mean and maximum PCB levels in the water table aquifer, and the maximum PCB level in the till aquifer, exceeded the proposed state MCL, the proposed federal MCL, the state GWQS, the proposed MCLG, the state SWQC, and the AWQC. The mean and maximum total PAH levels in the water table aquifer exceeded the state toxic effluent limit. The mean and maximum phenol concentrations in the water table aquifer exceeded the state GWQS while the maximum concentration exceeded the AWQC. The maximum nitrobenzene concentration in the water table aquifer exceeded the AWQC.

Several inorganics also exceeded ARARs and other guidance. The mean and maximum arsenic levels in the water table aquifer exceeded the AWQC. The maximum arsenic level in the water table also exceeded the federal and state MCL, the state GWQS, and the MCLG. The maximum cadmium, chromium, and lead concentrations in the water table exceeded their federal MCLs and MCLGs, their state MCLs and ground water quality standards, and their AWQC. The maximum mercury concentration in the water table exceeded the federal MCL and MCLG, the state MCL, and the state ground water quality standard. The mean and maximum nickel concentrations in the water table aquifer exceeded the AWQC for protection of human health. No inorganics were detected in the bedrock aquifer in concentrations which exceeded any ARARs or other guidance.

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5.2 QUANTITATIVE RISK CHARACTERIZATION

To quantitatively assess the risks to human health associated with present and future site and land use conditions, chronic average daily intakes (CDIs) are estimated for each exposure pathway using the estimated exposure point concentrations (see Section 2.0). CDIs are expressed as the amount of a chemical an individual may be exposed to per unit body weight per day, or mg/kg/day. A CDI is averaged over a lifetime for carcinogens and over the exposure period for noncarcinogens (USEPA 1989a). Appendix C presents the equations that were used to estimate CDIs for each pathway.

The estimated chronic daily intakes are then combined with health effects criteria (RfDs and cancer potency factors) to quantitatively estimate potential human health risks. For potential carcinogens, excess lifetime cancer risks are obtained by multiplying the CDI for the contaminant under consideration by its cancer potency factor. USEPA has implemented actions under Superfund associated with total cancer risks ranging from 10^{-4} to 10^{-6} (i.e., the incremental probability of developing cancer over a 70-year lifetime is one in 10,000 or one in 10,000,000, respectively, under the conditions of exposure). A risk level of 10^{-6}, representing an additional probability of one in 1,000,000 that an individual could contract cancer due to exposure to a potential carcinogen, is often used as a benchmark by regulatory agencies.

Potential risks for noncarcinogens are presented as the ratio of the CDI to the reference dose (RfD); i.e., CDI:RfD. The sum of all of the ratios of chemicals under consideration is called the hazard index. The hazard index is useful as a reference point for gauging the potential noncarcinogenic effects of environmental exposures to complex mixtures. In general, hazard indices which are less than one are not likely to be associated with any health risks, and are therefore less likely to be of concern than hazard indices greater than one. If the hazard index is greater than one, the compounds will be segregated according to their critical effects (target organs) and separate hazard indices will be derived for each effect (USEPA 1989a). A conclusion should not be categorically drawn, however, that all hazard indices less than one are "acceptable" or that hazard indices of greater than one are "unacceptable". This is a consequence of the perhaps one order of magnitude or greater uncertainty inherent in estimates of the RfD and CDI.

In accordance with USEPA's guidelines for evaluating the potential toxicity of complex mixtures (USEPA 1986c), it was assumed that the toxic effects of the site-related chemicals would be additive. Lifetime excess cancer risks and the CDI:RfD ratios were summed to indicate the potential risks associated with mixtures of potential carcinogens and noncarcinogens, respectively. In the absence of specific information on the toxicity of the mixture to be assessed or on similar mixtures, USEPA guidelines generally recommend assuming that the effects of different components on the mixtures are additive when affecting a particular organ or system. Synergistic or antagonistic interactions may be taken into account if there is specific information on particular combinations

of chemicals. In this risk assessment, it was assumed that the potential

effects of the site-related chemicals would be additive.

In this section, the CDIs of chemicals by potentially exposed individuals are

first calculated. To determine these CDIs, the assumptions concerning

chemical concentrations (exposure point concentrations), and exposure

conditions such as frequency and duration of exposure, are used together with

media intake parameters described in this section. For each exposure

scenario, two exposure cases are considered: an average case and, at the

request of USEPA Region II in accordance with USEPA (1989a) guidance, a

reasonable maximum exposure (RME) case. The average scenario is intended to

represent possible exposures to a typical individual; however, use of

inherently conservative health criteria will still result in an overestimation

of risk. The RME scenario is intended to place an upper-bound on the

potential risks by combining RME exposure estimates with upper bound health

criteria. The RME scenario incorporates the exposure parameter values

recommended in USEPA's (1989a) Superfund guidance to the extent possible given

that this project was well underway at the time the new USEPA (1989a) guidance

was released (e.g., see Section 3.3). For the average case scenario, average

values for exposure point concentrations (geometric means) are combined with

average values describing the extent, frequency, and duration of exposure.

For the RME case, maximum exposure point concentrations are combined with RME

parameter values specifically recommended by USEPA (1989a) describing the

extent, frequency and duration of exposure. Where such specific RME values

are recommended by USEPA (1989a), they have been used in this assessment; however, they have not been independently verified.

Chronic daily intakes, excess lifetime cancer risks, and CDI:RfD ratios for the chemicals of concern considered in this assessment, as well as the assumptions and procedures used to calculate these values, are presented for each of the selected exposure pathways in the subsections that follow.

5.2.1 Estimated Intakes and Risks Under Current Site and Land Use Conditions

5.2.1.1 Contact With On-Site Soil by Trespassers

This scenario evaluates potential exposures and risks through dermal contact and incidental ingestion of chemicals of concern in surface soils by site trespassers who may occasionally visit the site. For this scenario, it is assumed that teenagers would be most likely to occasionally trespass on the site given its general inaccessibility within an industrialized area. The assumptions used to estimate CDIs by teenagers via this exposure pathway are listed in Table 5-5 and the CDI equation is presented in Appendix C. The frequency of exposure estimates were derived by considering site-specific climate conditions. It is expected that during winter months, cold conditions and/or heavy clothing worn by individuals will limit the period during which exposure through dermal contact and incidental ingestion may occur. NOAA (1979) reports that over a 29 year period of record, the maximum daily temperatures were 32°F and below 18 days per year. It will be assumed

TABLE 5-5

ASSUMPTIONS USED FOR CALCULATING CHEMICAL INTAKES FROM
SURFACE SOIL BY TRESPASSERS AT THE SCP SITE

CURRENT SITE USE CONDITIONS

Parameter	Average Exposure Case	Reasonable Maximum Exposure Case
Frequency of Exposure	100 days/year	200 days/year
Age During Exposure Period	13-18 years	13-18 years
Years of Exposure	5 years	5 years
Average Body Weight Over Exposure Period	57 kg	57 kg
Soil Skin Contact Rate	430 mg/day	4,583 mg/day (a)
Dermal Absorption Factors:		
noncarcinogenic PAHs	0.03	0.05
carcinogenic PAHs	0.009	0.02
PCBs	0.07	0.07
bis(2-ethylhexyl)phthalate	0.003	0.03
other phthalates	0.05	0.05
mercury	0.1	0.1
other inorganics	0	0
volatile organics	0.10	0.10
chlorinated benzenes	0.10	0.10
dieldrin, aldrin	0.01	0.02
phenol	0.03	0.03
phenolic compounds	0.02	0.03
nitrobenzene	0.003	0.003
Quantity of Soil Ingested	13 mg/day	100 mg/day (a)
Relative Oral Absorption Factors (b):		
PCBs, PAHs, dieldrin, aldrin bis(2-ethylhexyl)phthalate	0.21	0.50
arsenic	0.8	0.8
others	1.0	1.0

NOTE: See text for discussion of exposure parameter values.

(a) The input parameter values listed have been based on guidance provided by USEPA (1989a).
(b) The relative absorption factors represent the difference in absorption from ingested soil versus from an ingested solvent or aqueous medium (the vehicles usually adminstered in studies from which cancer potency factors and reference doses are derived). Because arsenic's slope factor is based on an absorbed dose, the oral absorption factor in this table for arsenic represents the fraction of ingested arsenic in soil that can be absorbed from this matrix (USEPA 1984).

therefore that, under average exposure conditions, trespassers may come into contact with surface soil two days/week over the 347 (i.e., 365-18) days when temperatures were above 32°F (approximately 100 days/year), and that under RME conditions trespassers may contact soil four times each week over this period (i.e., approximately 200 days/year). The total exposure duration is assumed to be five years. Body weight estimates are derived from age- and sex-specific body weight information provided in USEPA (1985).

As mentioned above, for this pathway exposures due to both the absorption of chemicals from soil through the skin and the incidental ingestion of soil containing chemicals are estimated. To estimate the extent of dermal exposure to a chemical, the amount of chemical absorption and soil contact rates (based on the amount of soil accumulation and the area of exposed skin) need to be known. The uncertainty contained in these parameters is large and necessitates the use of approximations (McLaughlin 1984). For the average case, the soil contact rate was estimated based on an assumed 0.5 mg of soil contacted per cm^2 of exposed skin per exposure event (Lepow et al. 1975, CDHS 1987, Que Hee et al. 1975, Roels et al. 1980, Clement 1988). For the RME case, a soil contact rate of 1.45 mg/cm^2, as directed in USEPA (1989a) was used. It was assumed that exposure would be to the hands, and hands and arms for the average and RME exposure conditions, respectively. The 50th percentile surface areas [in accordance with USEPA (1989a) guidance] of the hands, and hands plus arms (in cm^2) were calculated based on data in USEPA (1985), to be 860 cm^2 and 3,161 cm^2, respectively.

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Precise, quantitative investigations into the amounts of chemicals that may be absorbed through the skin under conditions normally encountered in the environment (and assumed to occur for this assessment) are almost completely lacking. For a chemical to be absorbed by the skin from soil, it must be released from the soil matrix, pass through the stratum corneum, the epidermis, the dermis, and into the systemic circulation. In contrast, chemicals absorbed by the lung or gastrointestinal tract may pass through only two cells (Klassen 1986).

For the purposes of this assessment, the amount of exposure due to dermal absorption is evaluated by estimating the fraction of absorption of a chemical from contacted soil that may occur for the selected chemicals of concern. Where experimental data are available, chemical specific absorption factors are estimated for this assessment. Where data are not available, absorption factors are assumed based on recent USEPA guidance where available (USEPA 1989a), information from the scientific literature, analogy to other similar chemicals, and/or conservative default values.

As shown in Table 5-5, dermal absorption factors thus differ for many of the chemicals of concern being evaluated in this assessment. The dermal absorption factor of 0.07 used for PCBs was obtained from work by Wester et al. (1987) which showed that an average of 44.5% of the applied dose of pure PCBs (Aroclors 1242 and 1254) was absorbed. Since PCBs at the site are present in a soil matrix, they will be much less able to be absorbed than from a pure solution; thus the absorption from pure solution was adjusted for

(i.e., multiplied by) a relative bioavailability factor from the soil matrix based on Poiger and Schlatter's (1980) studies on 2,3,7,8-TCDD. The bioavailability factor is the ratio of the percent of applied dose absorbed through the skin into circulation from a soil matrix to the percent absorbed from a solvent matrix.

Dermal absorption factors for the PAHs were obtained from work by Yang et al. (1986a,b) which showed absorption percents for anthracene (a noncarcinogenic PAH) of 20.1% ± 6.4% absorbed in one day (Yang et al. 1986a) and for benzo(a)pyrene (a carcinogenic PAH) of 5.8% ± 2.2% absorbed in one day. These PAHs were also not applied in a soil matrix and thus, by analogy to 2,3,7,8-TCDD, were also adjusted for relative bioavailability. The results for anthracene were used for dermal absorption factors for noncarcinogenic PAHs, using the mean value for the average case and the mean plus two standard deviations for the plausible maximum case. In a similar manner, the results for benzo(a)pyrene were used for carcinogenic PAHs.

The dermal absorption factors for phenol (3%) and nitrobenzene (0.3%) were based on Feldman and Maibach (1970). Dermal absorption studies reported by Feldman and Maibach (1974) indicated that 7.8% ± 2.9% applied aldrin and 7.7% ± 3.2% dieldrin was absorbed from an acetone solution. The absorption factors for these two pesticides are based on these numbers, with adjustments for bioavailability based on Poiger and Schlatter (1980). The dermal absorption factors for other phenolic compounds were based on Roberts et al. (1977). The

values for bis(2-ethylhexyl)phthalate were based on analyogy to 2,3,7,8-TCDD data from Poiger and Schlatter (1980).

Dermal absorption of inorganics from contacted soil is assumed to be negligible based on observations from animal studies using concentrated solutions (Skog and Wahlberg 1964, Wahlberg 1968a,b). Absorption of mercury is assumed to be greater than for the other inorganics based on reports of mercury absorption after topical application (Bourgeois et al. 1986) and the tendency of mercury to be present in a vapor rather than purely solid phase (Hursh et al. 1989).

Insufficient experimental data were available to derive dermal absorption factors for the remaining chemicals of concern (e.g., other phthalates, chlorinated benzenes, volatile organics). These dermal absorption factors were assumed as shown in Table 5-5 by considering their relative tendencies to be absorbed compared to the other chemicals.

To evaluate exposures due to incidental soil ingestion, results from a recent study by Calabrese et al. (1989) were used for the average case while the ingestion rate specifically recommended by USEPA (1989a) was used for the RME case. Use of these estimates assumes that a trespasser would be involved in activities which would result in contact and incidental ingestion of soils.

Ingested chemicals present in a soil matrix may not be as readily absorbed through the gut due to their affinity for soil particles as chemicals ingested in a solvent or solution (i.e., the matrices usually administered in

experimental studies from which cancer potency factors and RfDs are derived).

The experimental studies of 2,3,7,8-TCDD conducted by Poiger and Schlatter (1980), McConnell et al. (1984), Lucier et al. (1986), Wendling et al. (1989), and van den Berg et al. (1986, 1987) suggest that the oral bioavailability of 2,3,7,8-TCDD from a fly ash or soil matrix may range from 7%-50% when compared to bioavailability from a solvent matrix (e.g., corn oil).[1] The average relative oral bioavailability of 2,3,7,8-TCDD from soil, based on available results from these published studies, is 21%. Because information on the relative bioavailability of persistent organic chemicals from a solvent versus a soil matrix is limited, relative oral absorption factors for PCBs, dieldrin, aldrin, PAHs, and bis(2-ethylhexyl)phthalate were estimated by analogy to 2,3,7,8-TCDD (i.e., 0.21 for the average case and 0.50 for the RME case). For the remaining chemicals of concern an oral absorption factor of 1.0 (a conservative default value) will be used. It should be noted that a recent study (Fries et al. 1989) has shown that the relative oral bioavailability of PCBs may be on the order of 82-84%. From this study, relative oral bioavailabilities can be estimated for only two PCB congeners. There are, however, over 200 PCB congeners that may be present in a sample of soil containing PCBs. Additionally, other similar studies on PCBs are not yet available. Thus although these results have not been incorporated into this assessment, future experimental results may provide additional insight into

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[1]It should be recognized that there is a difference between absorption and bioavailability. Absorption represents the extent to which a chemical is transported across or moves through the lining of the gut (or lung or skin). Bioavailability reflects the extent to which a chemical becomes available to be absorbed as well as absorption. Since most RfDs and slope factors are based on administered, not absorbed, doses, the relevant parameter to use in estimating exposures relates to the relative bioavailability from ingested soil versus an ingested aqueous or solvent medium, not absolute absorption.

PCB bioavailability. In addition, because arsenic's slope factor is based on an absorbed dose, the CDIs for arsenic (to evaluate potential carcinogenic effects) are calculated to reflect an absorbed dose using an absorption fraction of 0.8 (USEPA 1984).

Chronic daily intakes of the chemicals of concern, and the potential risks associated with this exposure pathway are summarized in Table 5-6. The formula for calculating the chronic daily intakes is present in Appendix C. Table 5-6 shows that the potential upper bound lifetime excess cancer risks to trespassers through dermal contact and incidental ingestion are 2×10^{-6} for the average case and 3×10^{-2} for the RME case. The average case risks are primarily due to exposures to carcinogenic PAHs and PCBs in surface soil. The RME case risk is primarily due to exposure to PCBs at their maximum detected surface soil concentration $(15,008 \text{ mg/kg})$[2], although for many other chemicals the risks exceed 1×10^{-6} (e.g., aldrin, carcinogenic PAHs, dieldrin, trichloroethylene). The RME case risk assumes, however, that a trespasser would repeatedly contact this specific location (or other non-sampled locations containing similar levels) over a 5-year period, a scenario which is likely to have a low probability of occurring. It should be recognized, however, that potential risks of developing skin cancer due to direct contact are not evaluated (due to the lack of detailed scientific data and USEPA-approved dose-response values).

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[2]The maximum concentration represents the sum of all the reported aroclor concentrations at a single sampling location treating non-detects as one-half the detection limit.

TABLE 5-6

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH DIRECT
SOIL CONTACT BY TRESPASSERS ON THE SCP SITE

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	SOIL CONCENTRATION (mg/kg) (a)		CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	0.044	57	1.06E-10	5.54E-06	1.70E+01 [B2]	1.81E-09	9.43E-05
Arsenic	8.1	60	2.89E-08	3.30E-06	2.00E+00 [A]	5.78E-08	6.59E-06
Benzene	0.090	54	1.73E-09	2.07E-05	2.90E-02 [A]	5.02E-11	5.99E-07
Bis(2-ethylhexyl)phthalate	34	281	4.69E-08	3.62E-05	1.40E-02 [B2]	6.57E-10	5.06E-07
Carcinogenic PAHs	8.9	51	2.02E-08	5.00E-06	1.15E+01 [B2]	2.32E-07	5.75E-05
Chloroform	0.044	18	8.46E-10	6.82E-06	6.10E-03 [B2]	5.16E-12	4.16E-08
1,1-Dichloroethane	0.072	65	1.38E-09	2.48E-05	9.10E-02 [B2]	1.26E-10	2.26E-06
1,2-Dichloroethane	0.060	10	1.15E-09	3.91E-06	9.10E-02 [B2]	1.05E-10	3.56E-07
1,1-Dichloroethylene	0.010	0.18	1.92E-10	6.90E-08	6.00E-01 [C]	1.15E-10	4.14E-08
Dieldrin	0.17	57	4.10E-10	5.54E-06	1.60E+01 [B2]	6.56E-09	8.87E-05
Isophorone	ND	ND	---	---	4.10E-03 [C]	---	---
Methylene chloride	0.14	2.4	2.69E-09	9.20E-07	7.50E-03 [B2]	2.02E-11	6.90E-09
PCBs	15	15,008	1.69E-07	3.82E-03	7.70E+00 [B2]	1.30E-06	2.90E-02
1,1,2,2-Tetrachloroethane	NC	0.48	---	1.84E-07	2.00E-01 [C]	---	3.68E-08
Tetrachloroethylene	0.93	4,290	1.79E-08	1.64E-03	5.10E-02 [B2]	9.12E-10	8.39E-05
Trichloroethylene	0.27	2,060	5.19E-09	7.90E-04	1.10E-02 [B2]	5.71E-11	8.69E-06
Vinyl chloride	ND	ND	---	---	2.30E+00 [A]	---	---
Total Excess Cancer Risk						2E-06	3E-02

(a) Based on shallow soil sampling results (0-2 feet).
(b) Chronic daily intakes for carcinogens were averaged over a 70-year lifetime (25,550 days).

NC = Not calculated because chemical was detected in only one sample.
ND = Not detected in shallow soil samples.

A

5-28

TABLE 5-6 (Continued)

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH DIRECT
SOIL CONTACT BY TRESPASSERS ON THE SCP SITE

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH NONCARCINOGENIC EFFECTS	SOIL CONCENTRATION (mg/kg) (a)		CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	0.044	57	1.49E-09	7.76E-05	3.00E-05	4.96E-05	2.59E+00
Antimony	3.8	16	2.37E-07	1.54E-05	4.00E-04	5.94E-04	3.85E-02
Arsenic	8.1	60	5.06E-07	5.77E-05	1.00E-03	5.06E-04	5.77E-02
Bis(2-ethylhexyl)phthalate	34	281	6.49E-07	5.06E-04	2.00E-02	3.25E-05	2.53E-02
Butyl benzyl phthalate	1.5	48	2.49E-07	1.53E-04	2.00E-01	1.24E-06	7.64E-04
Cadmium	6.1	95	3.81E-07	9.14E-05	1.00E-03	3.81E-04	9.14E-02
Chlorobenzene	0.13	336	3.50E-08	1.80E-03	2.00E-02	1.75E-06	9.02E-02
Chloroform	0.044	18	1.18E-08	9.55E-05	1.00E-02	1.18E-06	9.55E-03
2-Chloronaphthalene	0.17	122	1.28E-08	3.27E-04	NA	---	---
Chromium	79	721	4.91E-06	6.93E-04	5.00E-03	9.81E-04	1.39E-01
Cyanide	1.9	5.0	1.19E-07	4.81E-06	2.00E-02	5.94E-06	2.40E-04
1,2-Dichlorobenzene	0.54	47	1.45E-07	2.54E-04	9.00E-02	1.61E-06	2.82E-03
1,1-Dichloroethane	0.072	65	1.94E-08	3.47E-04	1.00E-01	1.94E-07	3.47E-03
1,1-Dichloroethylene	0.010	0.18	2.69E-09	9.66E-07	9.00E-03	2.99E-07	1.07E-04
1,2-trans-Dichloroethylene	0.009	0.24	2.42E-09	1.29E-06	2.00E-02	1.21E-07	6.44E-05
Dieldrin	0.17	57	5.74E-09	7.76E-05	5.00E-05	1.15E-04	1.55E+00
Di-n-butyl phthalate	3.1	71	5.14E-07	2.25E-04	·1.00E-01	5.14E-06	2.25E-03
Di-n-octyl phthalate	1.6	9.1	2.65E-07	2.88E-05	NA	---	---
2,4-Dimethylphenol	0.19	1.1	1.97E-08	2.51E-06	NA	---	---
Ethylbenzene	0.38	652	1.02E-07	3.50E-03	1.00E-01	1.02E-06	3.50E-02
Isophorone	ND	ND	---	---	2.00E-01	---	---
Lead	490	2,750	3.06E-05	2.64E-03	NA	---	---
Mercury	1.4	21	3.77E-07	1.14E-04	3.00E-04	1.26E-03	3.81E-01
Methylene chloride	0.14	2.4	3.77E-08	1.29E-05	6.00E-02	6.28E-07	2.15E-04
Methyl ethyl ketone	0.10	8.6	2.69E-08	4.62E-05	5.00E-02	5.38E-07	9.23E-04
Nickel	12	39	7.62E-07	3.75E-05	2.00E-02	3.81E-05	1.87E-03
Nitrobenzene	NC	78	---	8.56E-05	5.00E-04	---	1.71E-01
Noncarcinogenic PAHs	12	118	8.65E-07	3.16E-04	4.00E-01	2.16E-06	7.91E-04
PCBs	15	15,008	2.43E-06	5.35E-02	1.00E-04	2.43E-02	5.35E+02
Phenol	0.15	58	1.87E-08	1.33E-04	6.00E-01	3.11E-08	2.21E-04
Selenium	0.49	4.9	3.06E-08	4.71E-06	3.00E-03	1.02E-05	1.57E-03
Silver	1.1	19	6.87E-08	1.83E-05	3.00E-03	2.29E-05	6.09E-03
1,1,2,2-Tetrachloroethane	NC	0.48	---	2.58E-06	4.60E-04	---	5.60E-03
Tetrachloroethylene	0.93	4,290	2.50E-07	2.30E-02	1.00E-02	2.50E-05	2.30E+00
Toluene	0.74	3,380	1.99E-07	1.81E-02	3.00E-01	6.64E-07	6.05E-02
1,1,1-Trichloroethane	NC	1.2	---	6.44E-06	9.00E-02	---	7.16E-05
Trichloroethylene	0.27	2,060	7.27E-08	1.11E-02	7.35E-03	9.89E-06	1.50E+00
Xylenes	1.7	3,450	4.50E-07	1.85E-02	2.00E+00	2.25E-07	9.26E-03
Zinc	398	4,170	2.49E-05	4.01E-03	2.00E-01	1.24E-04	2.00E-02
Hazard Index						3E-02	5E+02

(a) Based on shallow soil sampling results (0-2 feet).
(b) Chronic daily intakes for noncarcinogens were averaged over 1,825 days for the average and maximum cases (5 yrs x 365 days/yr).

NA = Not available.
NC = Not calculated because chemical was detected in only one sample.
ND = Not detected in shallow soil samples.

For the noncarcinogens evaluated under this exposure scenario, the hazard index is less than one for the average exposure scenario indicating that noncarcinogenic health effects are not likely to result. For the RME case, the hazard index exceeds one. In accordance with USEPA (1989a) guidelines for a hazard index value greater than one, the chemicals were redivided based on their target organs and then hazard index values were recalculated for each target organ.

For the list of chemicals shown in Table 5-6, the most sensitive target organs for chronic exposure are as follows:

Liver

Aldrin
Bis(2-ethylhexyl)phthalate
Butyl benzyl phthalate
Chlorobenzene
Chloroform
Chromium (trivalent)
Dieldrin
Di-n-butylphthalate
1,1-Dichloroethylene
1,2-trans-Dichloroethylene
Ethylbenzene
Methylene chloride
Nitrobenzene
Noncarcinogenic PAHs
PCBs
Phenol
1,1,2,2,-Tetrachloroethane
Tetrachloroethylene
1,1,1-Trichloroethane
Trichloroethylene

Lung

Nickel

Heart

Antimony

Thyroid

Cyanide

Developmental

Methyl ethyl ketone
1,1,1-Trichloroethane

Lymph

Noncarcinogenic PAHs

Blood

Nickel
Nitrobenzene
1,1,2,2-Tetra-
   chloroethane
Zinc

Reproductive

PCBs

Kidney

Butyl benzyl
   phthalate
Cadmium
Chlorobenzene
1,2-Dichlorobenzene
1,1-Dichloroethane
1,1-Dichloroethylene
Ethylbenzene
Mercury
Nitrobenzene
Phenol

Central Nervous
System

1,1-Dichloroethylene
trans-1,2-Dichloro-
   ethylene
Dieldrin
1,1,2,2-Tetra-
   chloroethane
Toluene
Xylenes

Skin/Muscle

Arsenic
Selenium
Silver

A

5-30

For these target organ categories, the recalculated hazard index values still exceeded the threshold level of one for liver effects (due primarily to tetrachloroethylene, trichloroethylene, PCBs, dieldrin and aldrin), reproductive effects (due to PCBs) and central nervous system effects (due primarily to dieldrin). Thus, under this specific exposure scenario (trespassers on site), adverse effects to the liver, reproductive system and central nervous system could occur.

5.2.1.2 Inhalation of Volatilized Organics by Nearby Residents and Workers

Inhalation by Nearby Residents. To evaluate potential exposures and risks to nearby residents associated with inhalation of volatilized organics, it was assumed that a 70-kg individual would live at the nearest residence to the site continuously 365 days a year over a 30-year period for the RME case and over a 9-year period for the average case (USEPA 1989a). Average and RME inhalation rates of 13 m^3/day (USEPA 1985, NCRP 1984) to 30 m^3/day (USEPA 1989a), respectively, were used. The average inhalation rate is a weighted average across age groups based on USEPA (1985) and NCRP (1984). The maximum inhalation rate is based on the recommended RME value provided in recent USEPA guidance (USEPA 1989a). The air concentrations for this pathway were presented in Section 3.

Table 5-7 presents the chronic daily intakes and potential risks to the nearest resident via inhalation of volatilized organics. Chemicals of concern that were detected in soils and for which there are toxicity criteria have

TABLE 5-7

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INHALATION
OF VOLATILIZED ORGANICS FROM THE SCP SITE
BY NEARBY (OFF-SITE) RESIDENTS

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN RESIDENTIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	4.53E-11	1.08E-12	8.32E-12	1.70E+01 [B2]	1.84E-11	1.41E-10
Benzene	2.06E-07	4.92E-09	3.78E-08	2.90E-02 [A]	1.43E-10	1.10E-09
Bis(2-ethylhexyl)phthalate	4.49E-08	1.07E-09	8.25E-09	NA [B2]	--	--
Butyl benzyl phthalate	9.62E-09	2.30E-10	1.77E-09	NA [C]	--	--
Carcinogenic PAHs	4.61E-09	1.10E-10	8.47E-10	NA [B2]	--	--
Chloroform	1.21E-06	2.89E-08	2.22E-07	8.10E-02 [B2]	2.34E-09	1.80E-08
1,2-Dichloroethane	1.44E-07	3.44E-09	2.64E-08	9.10E-02 [B2]	3.13E-10	2.41E-09
1,1-Dichloroethylene	1.10E-06	2.63E-08	2.02E-07	1.20E+00 [C]	3.15E-08	2.42E-07
Dieldrin	1.36E-10	3.25E-12	2.50E-11	1.60E+01 [B2]	5.20E-11	4.00E-10
Methylene chloride	6.74E-06	1.61E-07	1.24E-06	1.40E-02 [B2]	2.25E-09	1.73E-08
PCBs	7.12E-08	1.70E-09	1.31E-08	NA [B2]	--	--
1,1,2,2-Tetrachloroethane	3.77E-09	9.00E-11	6.92E-10	2.00E-01 [C]	1.80E-11	1.38E-10
Tetrachloroethylene	6.21E-07	1.48E-08	1.14E-07	3.30E-03 [B2]	4.89E-11	3.76E-10
Trichloroethylene	6.41E-06	1.53E-07	1.18E-06	1.70E-02 [B2]	2.60E-09	2.00E-08
Vinyl chloride	3.60E-06	8.60E-08	6.61E-07	2.95E-01 [A]	2.54E-08	1.95E-07
Total Excess Lifetime Cancer Risk					6E-08	5E-07

CHEMICAL WITH NONCARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN RESIDENTIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Bis(2-ethylhexyl)phthalate	4.49E-08	8.34E-09	1.92E-08	NA	--	--
Butyl benzyl phthalate	9.62E-09	1.79E-09	4.12E-09	NA	--	--
Chlorobenzene	1.01E-07	1.88E-08	4.33E-08	5.00E-03	3.75E-06	8.66E-06
1,2-Dichlorobenzene	4.33E-08	8.04E-09	1.86E-08	4.00E-02	2.01E-07	4.64E-07
1,1-Dichloroethane	1.84E-06	3.42E-07	7.89E-07	1.00E-01	3.42E-06	7.89E-06
1,2-trans-Dichloroethylene	8.39E-07	1.56E-07	3.60E-07	NA	--	--
Di-n-butyl phthalate	9.32E-10	1.73E-10	3.99E-10	NA	--	--
Ethylbenzene	1.09E-07	2.02E-08	4.67E-08	NA	--	--
Methyl ethyl ketone	2.64E-07	4.90E-08	1.13E-07	9.00E-02	5.45E-07	1.26E-06
Methylene chloride	6.74E-06	1.25E-06	2.89E-06	8.60E-01	1.46E-06	3.36E-06
Noncarcinogenic PAHs	5.65E-07	1.05E-07	2.42E-07	NA	--	--
PCBs	7.12E-08	1.32E-08	3.05E-08	NA	--	--
Phenol	6.43E-08	1.19E-08	2.76E-08	NA	--	--
Toluene	1.65E-06	3.06E-07	7.07E-07	5.70E-01	5.38E-07	1.24E-06
1,1,1-Trichloroethane	6.67E-07	1.24E-07	2.86E-07	3.00E-01	4.13E-07	9.53E-07
Xylenes	1.47E-06	2.73E-07	6.30E-07	8.60E-02	3.17E-06	7.33E-06
Hazard Index					1E-05	3E-05

(a) Estimated air concentrations based on results of emission and dispersion modeling (see Section 3.3).
(b) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 9 or 30 yrs * 365 days/yr).

NA = Not available.

been evaluated. In accordance with directions presented in USEPA's guidance (1989a) and provided by USEPA's Environmental Criteria and Assessment Office (ECAO),[2] only inhalation route-specific toxicity criteria were used to evaluate this pathway. As shown in Table 5-7, for those chemicals with toxicity criteria for the inhalation route, this exposure scenario results in upper bound excess lifetime cancer risks of 6×10^{-8} to 5×10^{-7} for the average and RME cases, respectively. For both cases, the estimated risk is primarily due to inhalation of 1,1-dichloroethylene and vinyl chloride. For noncarcinogens, the hazard index values for the average and RME cases are less than one indicating that adverse noncarcinogenic effects are unlikely to occur. It should be noted, however, that risks were not quantitatively evaluated for several chemicals because of the lack of USEPA-approved slope factors or RfDs (e.g., for bis(2-ethylhexyl)phthalate, carcinogenic PAHs, PCBs, trans-1,2-dichloroethylene, and phenol). This could result in an underestimation of the hazard index values and excess lifetime cancer risks.

Inhalation by Nearby Workers. To evaluate potential inhalation exposures and risks to nearby (off-site) workers, it is assumed that workers would spend 8 hours employed near the site each day. It is also assumed that a 70-kg worker, engaging in light to moderate activities, will inhale over each 8-hour period, 7 m^3/day to 20 m^3/day for the average and RME exposure scenarios, respectively. The inhalation rate estimates are based on USEPA (1985). The frequency of exposure for these workers was assumed to be 100 days per year

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[2]P.F. Hurst. ECAO, Cincinnati, Ohio. Personal communication. January and February, 1990.

(two days/week, 50 weeks/year) for the average exposure case and 250 days per year (five days/week, 50 weeks/year) for the RME case. The durations of exposure were assumed to be 10 and 20 years for the average and RME cases, respectively. The air concentrations for this pathway were also presented in Section 3.

Table 5-8 summarizes the chronic daily intakes of the chemicals of concern, and the potential risks to nearby off-site workers associated with inhalation of volatilized organics. As shown in Table 5-8 for the average and RME exposure conditions evaluated, the upper bound excess lifetime cancer risks are 2×10^{-7} and 3×10^{-6}, respectively. These risks are predominantly due to inhalation of volatilized 1,1-dichloroethylene and vinyl chloride. For noncarcinogens, the hazard index is less than one for both the average and RME cases indicating adverse effects are unlikely to occur. As for the residential exposure scenario discussed above, these risks could be underestimated because several chemicals without USEPA-approved health criteria were not evaluated [e.g., bis(2-ethylhexyl)phthalate, carcinogenic PAHs, PCBs].

5.2.1.3 Inhalation of Suspended Soils by Nearby Residents and Workers

Inhalation by Nearby Residents. Fugitive dusts generated as a result of wind erosion may be transported in the air to nearby residences. To evaluate potential residential exposures via this inhalation pathway, the assumptions for inhalation of volatilized organics by the nearest resident were used. An

TABLE 5-8

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INHALATION OF VOLATILIZED ORGANICS FROM THE SCP SITE BY NEARBY (OFF-SITE) WORKERS

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN INDUSTRIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b) Average Case	Reasonable Maximum Case	SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK Average Case	Reasonable Maximum Case
Aldrin	9.54E-10	3.73E-12	5.33E-11	1.70E+01 [B2]	6.35E-11	9.07E-10
Benzene	4.34E-06	1.70E-08	2.43E-07	2.90E-02 [A]	4.93E-10	7.04E-09
Bis(2-ethylhexyl)phthalate	9.45E-07	3.70E-09	5.28E-08	NA [B2]	--	--
Butyl benzyl phthalate	2.02E-07	7.91E-10	1.13E-08	NA [C]	--	--
Carcinogenic PAHs	9.70E-08	3.80E-10	5.42E-09	NA [B2]	--	--
Chloroform	2.55E-05	9.98E-08	1.43E-06	8.10E-02 [B2]	8.08E-09	1.15E-07
1,2-Dichloroethane	3.04E-06	1.19E-08	1.70E-07	9.10E-02 [B2]	1.08E-09	1.55E-08
1,1-Dichloroethylene	2.31E-05	9.04E-08	1.29E-06	1.20E+00 [C]	1.08E-07	1.55E-06
Dieldrin	2.86E-09	1.12E-11	1.60E-10	1.60E+01 [B2]	1.79E-10	2.56E-09
Methylene chloride	1.42E-04	5.56E-07	7.94E-06	1.40E-02 [B2]	7.78E-09	1.11E-07
PCBs	1.50E-06	5.87E-09	8.39E-08	NA [B2]	--	--
1,1,2,2-Tetrachloroethane	7.94E-08	3.11E-10	4.44E-09	2.00E-01 [C]	6.22E-11	8.88E-10
Tetrachloroethylene	1.31E-05	5.13E-08	7.32E-07	3.30E-03 [B2]	1.69E-10	2.42E-09
Trichloroethylene	1.35E-04	5.28E-07	7.55E-06	1.70E-01 [B2]	8.98E-09	1.28E-07
Vinyl chloride	7.58E-05	2.97E-07	4.24E-06	2.95E-01 [A]	8.75E-08	1.25E-06
Total Excess Lifetime Cancer Risk					2E-07	3E-06

CHEMICAL WITH NONCARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN INDUSTRIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b) Average Case	Reasonable Maximum Case	REFERENCE DOSE (mg/kg/day)	HAZARD INDEX Average Case	Reasonable Maximum Case
Bis(2-ethylhexyl)phthalate	9.45E-07	2.59E-08	1.85E-07	NA	--	--
Butyl benzyl phthalate	2.02E-07	5.53E-09	3.95E-08	NA	--	--
Chlorobenzene	2.12E-06	5.81E-08	4.15E-07	5.00E-03	1.16E-05	8.30E-05
1,2-Dichlorobenzene	9.11E-07	2.50E-08	1.78E-07	4.00E-02	6.24E-07	4.46E-06
1,1-Dichloroethane	3.87E-05	1.06E-06	7.57E-06	1.00E-01	1.06E-05	7.57E-05
1,2-trans-Dichloroethylene	1.77E-05	4.85E-07	3.46E-06	NA	--	--
Di-n-butyl phthalate	1.96E-08	5.37E-10	3.84E-09	NA	--	--
Ethylbenzene	2.30E-06	6.30E-08	4.50E-07	NA	--	--
Methyl ethyl ketone	5.56E-06	1.52E-07	1.09E-06	9.00E-02	1.69E-06	1.21E-05
Methylene chloride	1.42E-04	3.89E-06	2.78E-05	8.60E-01	4.52E-06	3.23E-05
Noncarcinogenic PAHs	1.19E-05	3.26E-07	2.33E-06	NA	--	--
PCBs	1.50E-06	4.11E-08	2.94E-07	NA	--	--
Phenol	1.35E-06	3.70E-08	2.64E-07	NA	--	--
Toluene	3.47E-05	9.51E-07	6.79E-06	5.70E-01	1.67E-06	1.19E-05
1,1,1-Trichloroethane	1.40E-05	3.84E-07	2.74E-06	3.00E-01	1.28E-06	9.13E-06
Xylenes	3.10E-05	8.49E-07	6.07E-06	8.60E-02	9.88E-06	7.05E-05
Hazard Index					4E-05	3E-04

(a) Estimated air concentrations based on results of emission and dispersion modeling (see Section 3.3).
(b) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 days/yr).

NA = Not available.

individual was again assumed to live at the nearest residence for 365 days per year over a 9- or 30-year period. For the purposes of this assessment, it is conservatively assumed that chemicals present on inhaled suspended dust particles ($\leq$ 10 um in diameter) are 100% bioavailable in the lung. It is more likely that the suspended soil matrix may reduce the bioavailability of some chemicals, especially large organic molecules such as PCBs, below 100%. This is, however, considered to be an appropriate assumption for chemicals that affect the lung (e.g., chromium, PAHs). In addition, it was conservatively assumed that all chromium would be in the hexavalent form (Cr VI), which is carcinogenic when inhaled. For arsenic, because the slope factor is based on an absorbed dose, the percent absorption of inhaled arsenic was used in estimating its CDIs (for evaluating carcinogenic effects). Based on USEPA (1984), the absorption of arsenic in the lung was assumed to be 30%, while the pulmonary retention of inhaled dust was assumed to be 75% based on Schaum (1984). The resulting retention-absorption factor for arsenic was assumed to be 0.23 (i.e., 0.3*0.75).

Table 5-9 presents the chronic daily intakes and potential risks to the nearest resident via inhalation of suspended dusts. As shown in this table, the estimated upper bound excess lifetime cancer risks are 4×10^{-8} and 3×10^{-7} for the average and RME cases, respectively. These risks are primarily due to chromium. For noncarcinogens, the hazard index was less than one for both cases indicating adverse effects are unlikely to occur. Although it is conservative to assume that all inhaled chromium is in the hexavalent form, calculated risks may also be underestimated because several chemicals without

TABLE 5-9

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INHALATION
OF WIND ERODED SOIL FROM THE SCP SITE
BY NEARBY (OFF-SITE) RESIDENTS

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN RESIDENTIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	2.27E-11	5.42E-13	4.17E-12	1.70E+01 [B2]	9.21E-12	7.09E-11
Arsenic	4.18E-09	2.30E-11	1.77E-10	5.00E+01 [A]	1.15E-09	8.83E-09
Bis(2-ethylhexyl)phthalate	1.73E-08	4.13E-10	3.18E-09	NA [B2]	--	--
Butyl benzyl phthalate	7.95E-10	1.90E-11	1.46E-10	NA [C]	--	--
Cadmium	3.14E-09	7.50E-11	5.77E-10	6.10E+00 [B1]	4.57E-10	3.52E-09
Carcinogenic PAHs	4.36E-09	1.04E-10	8.01E-10	NA [B2]	--	--
Chromium	4.04E-08	9.65E-10	7.42E-09	4.10E+01 [A]	3.96E-08	3.04E-07
Dieldrin	8.76E-11	2.09E-12	1.61E-11	1.60E+01 [B2]	3.35E-11	2.57E-10
Nickel	6.29E-09	1.50E-10	1.16E-09	1.70E+00 [A]	2.55E-10	1.96E-09
PCBs	2.04E-09	4.87E-11	3.75E-10	NA [B2]	--	--
Total Excess Lifetime Cancer Risk					4E-08	3E-07

CHEMICAL WITH NONCARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN RESIDENTIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Antimony	1.96E-09	3.64E-10	8.39E-10	NA	--	--
Bis(2-ethylhexyl)phthalate	1.73E-08	3.21E-09	7.41E-09	NA	--	--
Butyl benzyl phthalate	7.95E-10	1.48E-10	3.41E-10	NA	--	--
2-Chloronaphthalene	8.96E-11	1.66E-11	3.84E-11	NA	--	--
Cyanide	9.53E-10	1.77E-10	4.08E-10	NA	--	--
1,2-Dichlorobenzene	2.80E-10	5.20E-11	1.20E-10	4.00E-02	1.30E-09	3.00E-09
2,4-Dimethylphenol	9.69E-11	1.80E-11	4.15E-11	NA	--	--
Di-n-butylphthalate	1.59E-09	2.95E-10	6.80E-10	NA	--	--
Di-n-octylphthalate	8.08E-10	1.50E-10	3.46E-10	NA	--	--
Lead	2.52E-07	4.69E-08	1.08E-07	NA	--	--
Mercury	7.21E-10	1.34E-10	3.09E-10	NA	--	--
Noncarcinogenic PAHs	4.62E-09	8.58E-10	1.98E-09	NA	--	--
PCBs	2.04E-09	3.79E-10	8.74E-10	NA	--	--
Phenol	7.47E-11	1.39E-11	3.20E-11	NA	--	--
Selenium	2.52E-10	4.69E-11	1.08E-10	1.00E-03	4.69E-08	1.08E-07
Silver	5.67E-10	1.05E-10	2.43E-10	NA	--	--
Zinc	2.05E-09	3.81E-10	8.79E-10	NA	--	--
Hazard Index					5E-08	1E-07

(a) Estimated air concentrations based on results of emission and dispersion modeling (See Section 3.3).
(b) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 9 or 30 yrs * 365 days/yr).

NA = Not available.

USEPA-approved health criteria were not evaluated (e.g., carcinogenic PAHs, PCBs, antimony, bis(2-ethylhexyl)phthalate, cyanide, 2,4-dimethylphenol, di-n-butylphthalate).

Inhalation by Nearby Workers. To evaluate potential exposures and risks via suspended dust inhalation by nearby off-site workers, the same assumptions used for inhalation of volatilized organics by workers were used. As for residents, it was also conservatively assumed that chemicals present on inhaled suspended dust particles ($\leq$ 10 um in diameter) are 100% bioavailable in the lung, and that all chromium would be in the hexavalent form. Similarly, to be consistent with its slope factor, the CDIs for arsenic (to evaluate potential carcinogenic effects) were calculated taking into account pulmonary absorption.

Table 5-10 presents the chronic daily intakes and the potential risks to nearby off-site workers via suspended dust inhalation. As can be seen from this table, the upper bound excess lifetime cancer risks are 1×10^{-7} and 2×10^{-6} for the average and RME cases, respectively. These risks are predominantly due to inhalation of chromium in suspended soil. It should be kept in mind that all chromium in soil was assumed to be in the hexavalent form, which is carcinogenic when inhaled. For noncarcinogens, adverse effects are unlikely to occur since the hazard index values are well below one. As noted for the other inhalation pathways, these risks may be underestimated due to the lack of USEPA-approved health criteria.

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TABLE 5-10

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INHALATION
BY WIND ERODED SOIL FROM THE SCP SITE
BY NEARBY (OFF-SITE) WORKERS

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN INDUSTRIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	4.77E-10	1.87E-12	2.67E-11	1.70E+01 [B2]	3.17E-11	4.53E-10
Arsenic	8.80E-08	7.92E-11	1.13E-09	5.00E+01 [A]	3.96E-09	5.66E-08
Bis(2-ethylhexyl)phthalate	3.64E-07	1.42E-09	2.04E-08	NA [B2]	--	--
Butyl benzyl phthalate	1.67E-08	6.54E-11	9.34E-10	NA [C]	--	--
Cadmium	6.61E-08	2.59E-10	3.70E-09	6.10E+00 [B1]	1.58E-09	2.25E-08
Carcinogenic PAHs	9.18E-08	3.59E-10	5.13E-09	NA [B2]	--	--
Chromium	8.51E-07	3.33E-09	4.76E-08	4.10E+01 [A]	1.37E-07	1.95E-06
Dieldrin	1.84E-09	7.20E-12	1.03E-10	1.60E+01 [B2]	1.15E-10	1.65E-09
Nickel	1.32E-07	5.17E-10	7.38E-09	1.70E+00 [A]	8.78E-10	1.25E-08
PCBs	4.28E-08	1.68E-10	2.39E-09	NA [B2]	--	--
Total Excess Lifetime Cancer Risk					1E-07	2E-06

CHEMICAL WITH NONCARCINOGENIC EFFECTS	OFF-SITE MAXIMUM AIR CONCENTRATION IN INDUSTRIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Antimony	4.12E-08	1.13E-09	8.06E-09	NA	--	--
Bis(2-ethylhexyl)phthalate	3.64E-07	9.97E-09	7.12E-08	NA	--	--
Butyl benzyl phthalate	1.67E-08	4.58E-10	3.27E-09	NA	--	--
2-Chloronaphthalene	1.89E-09	5.18E-11	3.70E-10	NA	--	--
Cyanide	2.01E-08	5.51E-10	3.93E-09	NA	--	--
1,2-Dichlorobenzene	5.89E-09	1.61E-10	1.15E-09	4.00E-02	4.03E-09	2.88E-08
2,4-Dimethylphenol	2.04E-08	5.59E-11	3.99E-10	NA	--	--
Di-n-butylphthalate	3.34E-08	9.15E-10	6.54E-09	NA	--	--
Di-n-octylphthalate	1.70E-08	4.66E-10	3.33E-09	NA	--	--
Lead	5.31E-06	1.45E-07	1.04E-06	NA	--	--
Mercury	1.52E-08	4.16E-10	2.97E-09	NA	--	--
Noncarcinogenic PAHs	9.72E-08	2.66E-09	1.90E-08	NA	--	--
PCBs	4.28E-08	1.17E-09	8.38E-09	NA	--	--
Phenol	1.57E-09	4.30E-11	3.07E-10	NA	--	--
Selenium	5.31E-09	1.45E-10	1.04E-09	1.00E-03	1.45E-07	1.04E-06
Silver	1.19E-08	3.26E-10	2.33E-09	NA	--	--
Zinc	4.31E-08	1.18E-09	8.43E-09	NA	--	--
Hazard Index					1E-07	1E-06

(a) Estimated air concentrations based on results of emission and dispersion modeling (see Section 3.3).
(b) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 days/yr).

NA = Not available.

5.2.2 Estimated Intakes and Risks Under Future Site and Land Use Conditions

5.2.2.1 Contact with Surface Soil by Future On-Site Workers

Potential exposures and risks to workers assumed to regularly work on the site in the future have been estimated. The assumptions used in estimating chemical intakes by future on-site workers are summarized in Table 5-11. These parameter values were derived in a similar manner as the current site use soil exposure scenarios (see Section 5.2.1.1). Under the average case scenario, future workers were assumed to be engaged in activities that could result in exposure to on-site soil two days per week for 50 weeks a year (100 days/year) for 10 years. Under the RME case, workers were assumed to be exposed five days per week for 50 weeks a year (250 days/year) for 20 years. Soil contact rates were calculated assuming exposure to the hands for the average case and the hands and forearms for the RME case, based on 50th percentile surface area estimates [as directed by USEPA (1989a)] of 990 cm^2 and 2,300 cm^2, respectively (USEPA 1985). With the exception of the duration and frequency of exposure, soil contact rates, and body weight, all other assumptions in Table 5-11 are identical to those listed in Table 5-5 for the current site use soil exposure scenario.

Table 5-12 presents the chronic daily intakes and the potential risks to future on-site workers via this exposure pathway. As can be seen from this table, the upper bound excess lifetime cancer risks are 3x10^{-6} and 9x10^{-2} for the average and RME cases, respectively. The risks are almost entirely due to

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TABLE 5-11

ASSUMPTIONS USED FOR CALCULATING CHEMICAL INTAKES FROM
SURFACE SOIL BY REGULAR WORKERS AT THE SCP SITE

FUTURE SITE USE CONDITIONS

Parameter	Average Exposure Case	Reasonable Maximum Exposure Case
Frequency of Exposure	100 days/year	250 days/year
Years of Exposure	10 years	20 years
Average Body Weight Over Exposure Period	70 kg	70 kg
Soil Skin Contact Rate	495 mg/day	3,335 mg/day (a)
Dermal Absorption Factors:		
noncarcinogenic PAHs	0.03	0.05
carcinogenic PAHs	0.009	0.02
PCBs	0.07	0.07
bis(2-ethylhexyl)phthalate	0.003	0.03
other phthalates	0.05	0.05
mercury	0.1	0.1
other inorganics	0	0
chlorinated benzenes	0.10	0.10
volatile organics	0.10	0.10
dieldrin, aldrin	0.01	0.02
phenol	0.03	0.03
phenolic compounds	0.02	0.03
nitrobenzene	0.003	0.003
Quantity of Soil Ingested	7 mg/day	100 mg/day (a)
Relative Oral Absorption Factors (b):		
PCBs, PAHs, dieldrin, aldrin bis(2-ethylhexyl)phthalate	0.21	0.50
arsenic	0.8	0.8
others	1.0	1.0

NOTE: See text for discussion of exposure parameter values.

(a) The input parameter values listed have been based on guidance provided by USEPA (1989a).
(b) The relative absorption factors represent the difference in absorption from ingested soil versus from an ingested solvent or aqueous medium (the vehicles usually adminstered in studies from which cancer potency factors and reference doses are derived). Because arsenic's slope factor is based on an absorbed dose, the oral absorption factor in this table for arsenic represents the fraction of ingested arsenic in soil that can be absorbed from this matrix (USEPA 1984).

TABLE 5-12

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH DIRECT
SOIL CONTACT BY ON-SITE WORKERS AT THE SCP SITE

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	SOIL CONCENTRATION (mg/kg) (a)		CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	0.044	57	1.58E-10	1.86E-05	1.70E+01 [B2]	2.69E-09	3.16E-04
Arsenic	8.1	60	2.54E-08	1.34E-05	2.00E+00 [A]	5.07E-08	2.68E-05
Benzene	0.090	54	2.84E-09	6.53E-05	2.90E-02 [A]	8.25E-11	1.89E-06
Bis(2-ethylhexyl)phthalate	34	281	5.55E-08	1.18E-04	1.40E-02 [B2]	7.77E-10	1.65E-06
Carcinogenic PAHs	8.9	51	2.96E-08	1.68E-05	1.15E+01 [B2]	3.40E-07	1.93E-04
Chloroform	0.044	18	1.39E-09	2.16E-05	6.10E-03 [B2]	8.48E-12	1.32E-07
1,1-Dichloroethane	0.072	65	2.27E-09	7.84E-05	9.10E-02 [B2]	2.07E-10	7.14E-06
1,2-Dichloroethane	0.060	10	1.90E-09	1.24E-05	9.10E-02 [B2]	1.72E-10	1.12E-06
1,1-Dichloroethylene	0.010	0.18	3.16E-10	2.18E-07	6.00E-01 [C]	1.90E-10	1.31E-07
Dieldrin	0.17	57	6.10E-10	1.86E-05	1.60E+01 [B2]	9.76E-09	2.97E-04
Isophorone	ND	ND	---	---	4.10E-03 [C]	---	---
Methylene chloride	0.14	2.4	4.42E-09	2.91E-06	7.50E-03 [B2]	3.32E-11	2.18E-08
PCBs	15	15,008	3.12E-07	1.19E-02	7.70E+00 [B2]	2.40E-06	8.75E-02
1,1,2,2-Tetrachloroethane	NC	0.48	---	5.82E-07	2.00E-01 [C]	---	1.16E-07
Tetrachloroethylene	0.93	4,290	2.94E-08	5.20E-03	5.10E-02 [B2]	1.50E-09	2.65E-04
Trichloroethylene	0.27	2,060	8.53E-09	2.50E-03	1.10E-02 [B2]	9.38E-11	2.75E-05
Vinyl chloride	ND	ND	---	---	2.30E+00 [A]	---	---
Total Excess Cancer Risk						3E-06	9E-02

(a) Based on shallow soil sampling results (0-2 feet).
(b) Chronic daily intakes for carcinogens were averaged over a 70-year lifetime (25,550 days).

NC = Not calculated because chemical was detected in only one sample.
ND = Not detected in shallow soil samples.

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TABLE 5-12 (Continued)

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH DIRECT
SOIL CONTACT BY ON-SITE WORKERS AT THE SCP SITE

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH NONCARCINOGENIC EFFECTS	SOIL CONCENTRATION (mg/kg) (a)		CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	0.044	57	1.11E-09	6.51E-05	3.00E-05	3.69E-05	2.17E+00
Antimony	3.8	16	1.04E-07	1.57E-05	4.00E-04	2.60E-04	3.91E-02
Arsenic	8.1	60	2.22E-07	5.87E-05	1.00E-03	2.22E-04	5.87E-02
Bis(2-ethylhexyl)phthalate	34	281	3.89E-07	4.13E-04	2.00E-02	1.94E-05	2.06E-02
Butyl benzyl phthalate	1.5	48	1.86E-07	1.26E-04	2.00E-01	9.32E-07	6.30E-04
Cadmium	6.1	95	1.67E-07	9.31E-05	1.00E-03	1.67E-04	9.31E-02
Chlorobenzene	0.13	336	2.87E-08	1.43E-03	2.00E-02	1.44E-06	7.13E-02
Chloroform	0.044	18	9.73E-09	7.55E-05	1.00E-02	9.73E-07	7.55E-03
2-Chloronaphthalene	0.17	122	1.09E-08	2.59E-04	NA	---	---
Chromium	79	721	2.15E-06	7.05E-04	5.00E-03	4.30E-04	1.41E-01
Cyanide	1.9	5.0	5.21E-08	4.89E-06	2.00E-02	2.60E-06	2.45E-04
1,2-Dichlorobenzene	0.54	47	1.19E-07	2.01E-04	9.00E-02	1.33E-06	2.23E-03
1,1-Dichloroethane	0.072	65	1.59E-08	2.74E-04	1.00E-01	1.59E-07	2.74E-03
1,1-Dichloroethylene	0.010	0.18	2.21E-09	7.64E-07	9.00E-03	2.46E-07	8.48E-05
1,2-trans-Dichloroethylene	0.009	0.24	1.99E-09	1.02E-06	2.00E-02	9.95E-08	5.09E-05
Dieldrin	0.17	57	4.27E-09	6.51E-05	5.00E-05	8.54E-05	1.30E+00
Di-n-butyl phthalate	3.1	71	3.85E-07	1.85E-04	1.00E-01	3.85E-06	1.85E-03
Di-n-octyl phthalate	1.6	9.1	1.99E-07	2.38E-05	NA	---	---
2,4-Dimethylphenol	0.19	1.1	1.26E-08	2.15E-06	NA	---	---
Ethylbenzene	0.38	652	8.40E-08	2.77E-03	1.00E-01	8.40E-07	2.77E-02
Isophorone	ND	ND	---	---	2.00E-01	---	---
Lead	490	2,750	1.34E-05	2.69E-03	NA	---	---
Mercury	1.4	21	3.10E-07	9.03E-05	3.00E-04	1.03E-03	3.01E-01
Methylene chloride	0.14	2.4	3.10E-08	1.02E-05	6.00E-02	5.16E-07	1.70E-04
Methyl ethyl ketone	0.10	8.6	2.21E-08	3.65E-05	5.00E-02	4.42E-07	7.30E-04
Nickel	12	39	3.34E-07	3.82E-05	2.00E-02	1.67E-05	1.91E-03
Nitrobenzene	NC	78	---	8.43E-05	5.00E-04	---	1.69E-01
Noncarcinogenic PAHs	12	118	7.36E-07	2.50E-04	4.00E-01	1.84E-06	6.25E-04
PCBs	15	15,008	2.18E-06	4.16E-02	1.00E-04	2.18E-02	4.16E+02
Phenol	0.15	58	1.28E-08	1.14E-04	6.00E-01	2.14E-08	1.90E-04
Selenium	0.49	4.9	1.34E-08	4.79E-06	3.00E-03	4.47E-06	1.60E-03
Silver	1.1	19	3.01E-08	1.86E-05	3.00E-03	1.00E-05	6.20E-03
1,1,2,2-Tetrachloroethane	NC	0.48	---	2.04E-06	4.60E-04	---	4.43E-03
Tetrachloroethylene	0.93	4,290	2.06E-07	1.82E-02	1.00E-02	2.06E-05	1.82E+00
Toluene	0.74	3,380	1.64E-07	1.43E-02	3.00E-01	5.45E-07	4.78E-02
1,1,1-Trichloroethane	NC	1.2	---	5.09E-06	9.00E-02	---	5.66E-05
Trichloroethylene	0.27	2,060	5.97E-08	8.74E-03	7.35E-03	8.12E-06	1.19E+00
Xylenes	1.7	3,450	3.69E-07	1.46E-02	2.00E+00	1.85E-07	7.32E-03
Zinc	398	4,170	1.09E-05	4.08E-03	2.00E-01	5.45E-05	2.04E-02
Hazard Index						2E-02	4E+02

(a) Based on shallow soil sampling results (0-2 feet).
(b) Chronic daily intakes for noncarcinogens were averaged over the period of exposure (i.e., 10 or 20 yrs * 365 days/yr).

NA = Not available.
NC = Not calculated because chemical was detected in only one sample.
ND = Not detected in shallow soil samples.

exposure to PCBs in surface soil. For the RME case, most of the selected chemicals of potential concern have risks which exceed 1×10^{-6}. The hazard index value for the RME case exceeds one; thus hazard index values were recalculated by target organ. The recalculated values were all less than one except for liver effects (due primarily to exposure to aldrin, dieldrin, PCBs, tetrachloroethylene and trichloroethylene), reproductive effects (due to PCBs) and central nervous system effects (due primarily to dieldrin), indicating that adverse effects to these organ systems might occur.

5.2.2.2 Contact with Subsurface Soil by Future On-Site Construction Workers

In the case of future development of the site, it is possible that some workers will be exposed to subsurface soils during excavations and construction. For this assessment, it was assumed that a worker would be exposed to subsurface soils in the saturated fill (5-6 feet deep) during construction of a basement foundation. It was further assumed that the foundation job would result in subsurface soil exposures 5 days/week for 4 weeks. The worker would only be exposed during this period and was assumed not to be involved in another foundation construction on site. Because of its short duration (i.e., less than one year), this scenario would result in subchronic (i.e., short-term) exposures. To evaluate potential noncarcinogenic effects of these exposures, subchronic rather than chronic RfDs are used (USEPA 1989a). Although USEPA has not yet adopted such subchronic RfDs on its IRIS database, the subchronic RfDs from USEPA Health

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Effects Assessments (HEAs) shown in Table 4-1 will be used to evaluate this pathway.

The assumptions used in estimating chemical intakes by future on-site workers to subsurface soil are summarized in Table 5-13. With the exception of the duration and frequency of exposure, all assumptions in Table 5-13 are identical to those listed in Table 5-2 for the current use soil exposure scenario.

Table 5-14 presents the chronic daily intakes and the potential risks to future site construction workers via this exposure pathway. As can be seen from this table, the upper bound excess lifetime cancer risks are 2×10^{-8} for the average case and 2×10^{-5} for the RME case, due primarily to exposure to PCBs and carcinogenic PAHs in the saturated fill (5-6 feet deep). The hazard index values are less than one indicating adverse effects are unlikely to occur for this construction scenario.

5.2.2.3 Inhalation of Volatilized Organics by Future On-Site Workers

The assumptions used to estimate potential exposures of future on-site workers from inhalation of volatilized organics are identical to those used for current exposures to nearby off-site workers (see Section 5.2.1.2), except for the estimated air concentrations. The on-site air concentrations used for this scenario were presented in Section 3.

TABLE 5-13

ASSUMPTIONS USED FOR CALCULATING CHEMICAL INTAKES FROM
SUBSURFACE SOIL BY FUTURE CONSTRUCTION WORKERS AT THE SCP SITE

CURRENT SITE USE CONDITIONS

Parameter	Average Exposure Case	Reasonable Maximum Exposure Case
Frequency of Exposure	20 days/year	20 days/year
Years of Exposure	1 year	1 year
Average Body Weight Over Exposure Period	70 kg	70 kg
Soil Skin Contact Rate	495 mg/day	3,335 mg/day (a)
Dermal Absorption Factors:		
noncarcinogenic PAHs	0.03	0.05
carcinogenic PAHs	0.009	0.02
PCBs	0.07	0.07
bis(2-ethylhexyl)phthalate	0.003	0.03
other phthalates	0.05	0.05
mercury	0.1	0.1
other inorganics	0	0
chlorinated benzenes	0.10	0.10
volatile organics	0.10	0.10
dieldrin, aldrin	0.01	0.02
phenol	0.03	0.03
phenolic compounds	0.02	0.03
nitrobenzene	0.003	0.03
Quantity of Soil Ingested	7 mg/day	100 mg/day (a)
Relative Oral Absorption Factors (b):		
PCBs, PAHs, dieldrin, aldrin bis(2-ethylhexyl)phthalate	0.21	0.50
arsenic	0.8	0.8
others	1.0	1.0

NOTE: See text for discussion of exposure parameter values.

(a) The input parameter values listed have been based on guidance provided by USEPA (1989a).
(b) The relative absorption factors represent the difference in absorption from ingested soil versus from an ingested solvent or aqueous medium (the vehicles usually adminstered in studies from which cancer potency factors and reference doses are derived). Because arsenic's slope factor is based on an absorbed dose, the oral absorption factor in this table for arsenic represents the fraction of ingested arsenic in soil that can be absorbed from this matrix (USEPA 1984).

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TABLE 5-14

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH DIRECT
SOIL CONTACT BY CONSTRUCTION WORKERS AT THE SCP SITE

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	SOIL CONCENTRATION (mg/kg) (a)		CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]		EXCESS UPPER BOUND LIFETIME CANCER RISK	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case			Average Case	Reasonable Maximum Case
Aldrin	NC	1.2	---	1.57E-09	1.70E+01	[B2]	---	2.66E-08
Arsenic	7.8	62	4.88E-10	5.55E-08	2.00E+00	[A]	9.77E-10	1.11E-07
Benzene	0.62	52	3.92E-10	2.54E-07	2.90E-02	[A]	1.14E-11	7.35E-09
Bis(2-ethylhexyl)phthalate	14	381	4.76E-10	6.39E-07	1.40E-02	[B2]	6.66E-12	8.95E-09
Carcinogenic PAHs	7.5	653	4.98E-10	8.52E-07	1.15E+01	[B2]	5.73E-09	9.80E-08
Chloroform	0.26	379	1.64E-10	1.84E-06	6.10E-03	[B2]	1.00E-12	1.12E-08
1,1-Dichloroethane	0.46	179	2.91E-10	8.68E-07	9.10E-02	[B2]	2.64E-11	7.90E-08
1,2-Dichloroethane	0.41	290	2.59E-10	1.41E-06	9.10E-02	[B2]	2.36E-11	1.28E-07
1,1-Dichloroethylene	ND	ND	---	---	6.00E-01	[C]	---	---
Dieldrin	0.023	0.94	1.65E-12	1.23E-09	1.60E+01	[B2]	2.64E-11	1.96E-08
Isophorone	ND	ND	---	---	4.10E-03	[C]	---	---
Methylene chloride	0.57	15	3.60E-10	7.22E-08	7.50E-03	[B2]	2.70E-12	5.42E-10
PCBs	4.3	351	1.72E-09	1.11E-06	7.70E+00	[B2]	1.32E-08	8.57E-06
1,1,2,2-Tetrachloroethane	NC	0.70	---	3.39E-09	2.00E-01	[C]	---	6.79E-10
Tetrachloroethylene	2.8	1,690	1.74E-09	8.19E-06	5.10E-02	[B2]	8.89E-11	4.18E-07
Trichloroethylene	0.86	1,670	5.43E-10	8.10E-06	1.10E-02	[B2]	5.98E-12	8.91E-08
Vinyl chloride	NC	0.029	---	1.41E-10	2.30E+00	[A]	---	3.23E-10
Total Excess Cancer Risk							2E-08	2E-05

(a) Soil concentrations based on medium depth soil samples (5-6 feet) in saturated fill.
(b) Chronic daily intakes for carcinogens were averaged over a 70-year lifetime (25,550 days).

NC = Not calculated because chemical was detected in only one sample.
ND = Not detected in medium depth soil samples.

TABLE 5-14 (Continued)

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH DIRECT
SOIL CONTACT BY CONSTRUCTION WORKERS AT THE SCP SITE

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH NONCARCINOGENIC EFFECTS	SOIL CONCENTRATION (mg/kg) (a)		CHRONIC DAILY INTAKE (mg/kg/day) (b)		SUBCHRONIC REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	NC	1.2	---	1.10E-07	3.00E-05	---	3.65E-03
Antimony	4.5	38	2.47E-08	2.97E-06	4.00E-04	6.16E-05	7.44E-03
Arsenic	7.8	62	4.27E-08	4.85E-06	1.00E-03	4.27E-05	4.85E-03
Bis(2-ethylhexyl)phthalate	14	381	3.33E-08	4.48E-05	2.00E-02	1.67E-06	2.24E-03
Butyl benzyl phthalate	2.0	74	4.97E-08	1.54E-05	2.00E+00	2.49E-08	7.68E-06
Cadmium	3.9	26	2.14E-08	2.04E-06	1.00E-03 (c)	2.14E-05	2.04E-03
Chlorobenzene	0.89	258	3.94E-08	8.75E-05	2.00E-01	1.97E-07	4.38E-04
Chloroform	0.26	379	1.15E-08	1.29E-04	1.00E-02	1.15E-06	1.29E-02
2-Chloronaphthalene	0.28	18	4.79E-09	3.80E-06	NA	---	---
Chromium	57	542	3.12E-07	4.24E-05	5.00E-03 (c)	6.25E-05	8.49E-03
Cyanide	0.001	0.032	5.48E-12	2.50E-09	2.00E-02	2.74E-10	1.25E-07
1,2-Dichlorobenzene	0.50	385	2.21E-08	1.31E-04	9.00E-01	2.46E-08	1.45E-04
1,1-Dichloroethane	0.46	179	2.03E-08	6.07E-05	1.00E+00	2.03E-08	6.07E-05
1,1-Dichloroethylene	ND	ND	---	---	9.00E-03	---	---
1,2-trans-Dichloroethylene	0.29	512	1.27E-08	1.74E-04	2.00E-02 (c)	6.37E-07	8.69E-03
Dieldrin	0.023	0.94	1.16E-10	8.59E-08	5.00E-05	2.31E-06	1.72E-03
Di-n-butyl phthalate	1.8	98	4.47E-08	2.05E-05	1.00E+00	4.47E-08	2.05E-05
Di-n-octyl phthalate	1.2	20	2.98E-08	4.07E-06	NA	---	---
2,4-Dimethylphenol	0.38	11	5.03E-09	1.69E-06	NA	---	---
Ethylbenzene	4.3	529	1.90E-07	1.80E-04	1.00E+00	1.90E-07	1.80E-04
Isophorone	ND	ND	---	---	2.00E+00	---	---
Lead	271	2,810	1.48E-06	2.20E-04	NA	---	---
Mercury	0.75	14	3.32E-08	4.61E-06	3.00E-04	1.11E-04	1.54E-02
Methylene chloride	0.57	15	2.52E-08	5.06E-06	6.00E-02	4.20E-07	8.43E-05
Methyl ethyl ketone	1.3	795	5.75E-08	2.70E-04	5.00E-01	1.15E-07	5.40E-04
Nickel	29	116	1.59E-07	9.08E-06	2.00E-02	7.95E-06	4.54E-04
Noncarcinogenic PAHs	9.8	1,264	1.25E-07	2.14E-04	4.00E-01	3.11E-07	5.36E-04
PCBs	4.3	351	1.20E-07	7.79E-05	1.00E-04 (c)	1.20E-03	7.79E-01
Phenol	0.41	790	7.01E-09	1.24E-04	6.00E-01	1.17E-08	2.06E-04
Selenium	0.45	2.1	2.47E-09	1.64E-07	4.00E-03	6.16E-07	4.11E-05
Silver	NC	40	---	3.13E-06	3.00E-03 (c)	---	1.04E-03
1,1,2,2-Tetrachloroethane	NC	0.70	---	2.38E-07	4.60E-04 (c)	---	5.16E-04
Tetrachloroethylene	2.8	1,690	1.22E-07	5.73E-04	1.00E-01	1.22E-06	5.73E-03
Toluene	16	2,270	6.93E-07	7.70E-04	4.00E-01	1.73E-06	1.93E-03
1,1,1-Trichloroethane	0.47	1,770	2.08E-08	6.01E-04	9.00E-01	2.31E-08	6.67E-04
Trichloroethylene	0.86	1,670	3.80E-08	5.67E-04	7.35E-03 (c)	5.17E-06	7.71E-02
Xylenes	23	2,290	1.02E-06	7.77E-04	4.00E+00	2.54E-07	1.94E-04
Zinc	338	1,870	1.85E-06	1.46E-04	2.00E-01	9.26E-06	7.32E-04
Total Hazard Index						2E-03	9E-01

(a) Soil concentrations based on medium depth soil samples (5-6 feet) in saturated fill.
(b) Chronic daily intakes for noncarcinogens were averaged over 365 days (1 yr x 365 days/yr).
(c) In the absence of a subchronic reference dose, the chronic reference dose was used.

NA = Not available.
NC = Not calculated because chemical was detected in only one sample.
ND = Not detected in medium depth soil samples.

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Table 5-15 presents the chronic daily intakes and the potential risks to future site workers via this exposure pathway. As can be seen from this table, the upper bound excess lifetime cancer risks are 3×10^{-6} for the average case and 4×10^{-5} for the RME case. These risks are primarily due to inhalation of volatilized chloroform, 1,1-dichloroethylene, methylene chloride, trichloroethylene, and vinyl chloride. The hazard index values are less than one indicating that noncarcinogenic effects are unlikely to occur.

5.2.2.4   Inhalation of Suspended Soils by Future On-Site Workers

The assumptions used to estimate potential exposures of future on-site workers from inhalation of suspended soils are identical to those used for current exposures to nearby off-site workers (see Section 5.2.1.3), except for the estimated air concentrations (see Section 3).

Table 5-16 presents the chronic daily intakes and the potential risks to future site workers via this exposure pathway. As can be seen from this table, the upper bound excess lifetime cancer risks are 2×10^{-6} and 3×10^{-5} for the average and RME cases, respectively. These risks are primarily associated with inhaling chromium on suspended soil particles. The chromium risks are, however, very conservative since all chromium is assumed to be in the hexavalent form (i.e., carcinogenic). The hazard index values are less than one indicating that noncarcinogenic effects are unlikely to occur.

TABLE 5-15

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INHALATION
OF VOLATILIZED ORGANICS FROM THE SCP SITE
BY FUTURE (ON-SITE) WORKERS

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	ON-SITE AIR CONCENTRATION (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	1.29E-08	5.05E-11	7.21E-10	1.70E+01 [B2]	8.58E-10	1.23E-08
Benzene	5.85E-05	2.29E-07	3.27E-06	2.90E-02 [A]	6.64E-09	9.49E-08
Bis(2-ethylhexyl)phthalate	1.27E-05	4.97E-08	7.10E-07	NA [B2]	--	--
Butyl benzyl phthalate	2.73E-06	1.07E-08	1.53E-07	NA [C]	--	--
Carcinogenic PAHs	1.31E-06	5.13E-09	7.32E-08	NA [B2]	--	--
Chloroform	3.43E-04	1.34E-06	1.92E-05	8.10E-02 [B2]	1.09E-07	1.55E-06
1,2-Dichloroethane	4.10E-05	1.60E-07	2.29E-06	9.10E-02 [B2]	1.46E-08	2.09E-07
1,1-Dichloroethylene	3.11E-04	1.22E-06	1.74E-05	1.20E+00 [C]	1.46E-06	2.09E-05
Dieldrin	3.86E-08	1.51E-10	2.16E-09	1.60E+01 [B2]	2.42E-09	3.45E-08
Methylene chloride	1.91E-03	7.48E-06	1.07E-04	1.40E-02 [B2]	1.05E-07	1.50E-06
PCBs	2.02E-05	7.91E-08	1.13E-06	NA [B2]	--	--
1,1,2,2-Tetrachloroethane	1.07E-06	4.19E-09	5.98E-08	2.00E-01 [C]	8.38E-10	1.20E-08
Tetrachloroethylene	1.76E-04	6.89E-07	9.84E-06	3.30E-03 [B2]	2.27E-09	3.25E-08
Trichloroethylene	1.82E-03	7.12E-06	1.02E-04	1.70E-02 [B2]	1.21E-07	1.73E-06
Vinyl chloride	1.02E-03	3.99E-06	5.70E-05	2.95E-01 [A]	1.18E-06	1.68E-05
Total Excess Cancer Risk					3E-06	4E-05

CHEMICAL WITH NONCARCINOGENIC EFFECTS	ON-SITE AIR CONCENTRATION (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Bis(2-ethylhexyl)phthalate	1.27E-05	3.48E-07	2.49E-06	NA	--	--
Butyl benzyl phthalate	2.73E-06	7.48E-08	5.34E-07	NA	--	--
Chlorobenzene	2.86E-05	7.84E-07	5.60E-06	5.00E-03	1.57E-04	1.12E-03
1,2-Dichlorobenzene	1.23E-05	3.37E-07	2.41E-06	4.00E-02	8.42E-06	6.02E-05
1,1-Dichloroethane	5.22E-04	1.43E-05	1.02E-04	1.00E-01	1.43E-04	1.02E-03
1,2-trans-Dichloroethylene	2.38E-04	6.52E-06	4.66E-05	NA	--	--
Di-n-butyl phthalate	2.64E-07	7.23E-09	5.17E-08	NA	--	--
Ethylbenzene	3.10E-05	8.49E-07	6.07E-06	NA	--	--
Methyl ethyl ketone	7.49E-05	2.05E-06	1.47E-05	9.00E-02	2.28E-05	1.63E-04
Methylene chloride	1.91E-03	5.23E-05	3.74E-04	8.60E-01	6.08E-05	4.35E-04
Noncarcinogenic PAHs	1.60E-04	4.38E-06	3.13E-05	NA	--	--
PCBs	2.02E-05	5.53E-07	3.95E-06	NA	--	--
Phenol	1.82E-05	4.99E-07	3.56E-06	NA	--	--
Toluene	4.68E-04	1.28E-05	9.16E-05	5.70E-01	2.25E-05	1.61E-04
1,1,1-Trichloroethane	1.89E-04	5.18E-06	3.70E-05	3.00E-01	1.73E-05	1.23E-04
Xylenes	4.18E-04	1.15E-05	8.18E-05	8.60E-02	1.33E-04	9.51E-04
Hazard Index					6E-04	4E-03

(a) Estimated air concentrations based on results of emission and dispersion modeling (see Section 3.3).
(b) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 days/yr).

NA = Not available.

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TABLE 5-16

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INHALATION
OF WIND ERODED SOIL FROM THE SCP SITE
BY FUTURE (ON-SITE) WORKERS

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	ON-SITE MAXIMUM AIR CONCENTRATION IN INDUSTRIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Aldrin	6.43E-09	2.52E-11	3.60E-10	1.70E+01 [B2]	4.28E-10	6.11E-09
Arsenic	1.19E-06	1.07E-09	1.53E-08	5.00E+01 [A]	5.36E-08	7.65E-07
Bis(2-ethylhexyl)phthalate	4.91E-06	1.92E-08	2.75E-07	NA [B2]	--	--
Butyl benzyl phthalate	2.26E-07	8.85E-10	1.26E-08	NA [C]	--	--
Cadmium	8.92E-07	3.49E-09	4.99E-08	6.10E+00 [B1]	2.13E-08	3.04E-07
Carcinogenic PAHs	1.24E-06	4.85E-09	6.93E-08	NA [B2]	--	--
Chromium	1.15E-05	4.50E-08	6.43E-07	4.10E+01 [A]	1.85E-06	2.64E-05
Dieldrin	2.48E-08	9.71E-11	1.39E-09	1.60E+01 [B2]	1.55E-09	2.22E-08
Nickel	1.78E-06	6.97E-09	9.95E-08	1.70E+00 [A]	1.18E-08	1.69E-07
PCBs	5.77E-07	2.26E-09	3.23E-08	NA [B2]	--	--
Total Excess Cancer Risk					2E-06	3E-05

CHEMICAL WITH NONCARCINOGENIC EFFECTS	ON-SITE MAXIMUM AIR CONCENTRATION IN INDUSTRIAL AREA (mg/m3) (a)	CHRONIC DAILY INTAKE (mg/kg/day) (b)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
		Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Antimony	5.55E-07	1.52E-08	1.09E-07	NA	--	--
Bis(2-ethylhexyl)phthalate	4.91E-06	1.35E-07	9.61E-07	NA	--	--
Butyl benzyl phthalate	2.26E-07	6.19E-09	4.42E-08	NA	--	--
2-Chloronaphthalene	2.54E-08	6.96E-10	4.97E-09	NA	--	--
Cyanide	2.70E-07	7.40E-09	5.28E-08	NA	--	--
1,2-Dichlorobenzene	7.94E-08	2.18E-09	1.55E-08	4.00E-02	5.44E-08	3.88E-07
2,4-Dimethylphenol	2.75E-08	7.53E-10	5.38E-09	NA	--	--
Di-n-butyl phthalate	4.50E-07	1.23E-08	8.81E-08	NA	--	--
Di-n-octyl phthalate	2.29E-07	6.27E-09	4.48E-08	NA	--	--
Lead	7.16E-05	1.96E-06	1.40E-05	NA	--	--
Mercury	2.05E-07	5.62E-09	4.01E-08	NA	--	--
Noncarcinogenic PAHs	1.31E-06	3.59E-08	2.56E-07	NA	--	--
PCBs	5.77E-07	1.58E-08	1.13E-07	NA	--	--
Phenol	2.12E-08	5.81E-10	4.15E-09	NA	--	--
Selenium	7.16E-08	1.96E-09	1.40E-08	1.00E-03	1.96E-06	1.40E-05
Silver	1.61E-07	4.41E-09	3.15E-08	NA	--	--
Zinc	5.82E-07	1.59E-08	1.14E-07	NA	--	--
Hazard Index					2E-06	1E-05

(a) Estimated air concentrations based on results of emission and dispersion modeling (see Section 3.3).
(b) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 days/yr).

NA = Not available.

5.2.2.5 Ingestion of Ground Water by Future On-Site Workers

For this pathway, it was assumed that a future on-site worker would obtain
drinking water from an on-site ground water well. The water table, till, and
bedrock aquifers are each evaluated separately. It should, however, be
recognized that among these aquifers, only the bedrock aquifer is currently
used as a drinking water supply. Although the water table and till aquifers
are considered unlikely to be suitable for a water supply (e.g., for example,
the till aquifer is tidally influenced and of too low permeability to be used
for a water supply), these aquifers are conservatively evaluated assuming they
are used for drinking water. Furthermore, available ground water data
indicate that contaminants in these two shallower aquifers are migrating to
the deeper bedrock aquifer.

The assumptions used to evaluate exposures to the chemicals of concern through
ingestion of ground water were that a 70-kg future on-site worker ingests one
liter of ground water 250 days/year for 10 years (average case) or 20 years
(RME case). The drinking water intake rate of one liter per day was
determined based on an adult daily drinking rate of two l/day (USEPA 1989a)
adjusted for the 8 hours per day spent on site. (Although some workers may
ingest more than one liter of liquid during heavy activity, it was assumed
that liquid sources other than ground water would also be available for use
[e.g., soda, juice]). The same ingestion rate and frequency of exposure
assumptions were used for both the average and RME scenarios. The

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concentrations used for this pathway were the geometric means (used for the average case) and the maximums (used for the RME case).

Table 5-17 presents the chronic daily intakes and the potential risks to a future worker via ingestion of water table aquifer ground water. As shown in this table, this exposure scenario results in upper bound excess lifetime cancer risks of $6x10^{-4}$ and $5x10^{-1}$ for the average and RME cases, respectively. The excess lifetime cancer risks exceed $1x10^{-6}$ for every chemical carcinogen for the RME case, and for most chemicals for the average case. It should be noted that potential risks due to water uses other than drinking (e.g., inhalation of volatile organics released into indoor air from showering, laundering, etc.) may also be associated with risks of a similar order of magnitude.

The hazard index values for the chemicals in the water table aquifer with noncarcinogenic effects were greater than one for both the average and RME cases. When recalculated by target organ categories (in accordance with USEPA risk assessment guidance), the RME case hazard index values were still greater than one for those chemicals that may affect the liver (due primarily to chlorobenzene, chloroform, trans-1,2-dichloroethylene, methylene chloride, nitrobenzene, PCBs, 1,1,2,2-tetrachloroethane, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene), the kidney (due primarily to chlorobenzene, 1,1-dichloroethane, nitrobenzene), the central nervous system (due to trans-1,2-dichloroethylene, toluene, 1,1,2,2-tetrachloroethane), the blood (due to nitrobenzene, 1,1,2,2-tetrachloroethane), development (due to

TABLE 5-17

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INGESTION
OF GROUND WATER BY FUTURE (ON-SITE) WORKERS
WATER TABLE AQUIFER

CHEMICAL WITH CARCINOGENIC EFFECTS	WATER CONCENTRATION (ug/l)		CHRONIC DAILY INTAKE (mg/kg/day) (a)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Arsenic	31	3,100	4.29E-05	8.67E-03	2.00E+00 [A]	8.58E-05	1.72E-02
Benzene	318	7,270	4.45E-04	2.03E-02	2.90E-02 [A]	1.29E-05	5.89E-04
Bis(2-ethylhexyl)phthalate	17	654	2.39E-05	1.83E-03	1.40E-02 [B2]	3.35E-07	2.56E-05
Carcinogenic PAHs	6.8	380	9.51E-06	1.06E-03	1.15E+01 [B2]	1.09E-04	1.21E-02
Chloroform	38	614,000	5.33E-05	1.72E+00	6.10E-03 [B2]	3.25E-07	1.04E-02
1,1-Dichloroethane	87	11,700	1.21E-04	3.27E-02	9.10E-02 [B2]	1.10E-05	2.97E-03
1,2-Dichloroethane	34	473,000	4.74E-05	1.32E+00	9.10E-02 [B2]	4.31E-06	1.13E-01
1,1-Dichloroethylene	NC	32	--	8.95E-05	6.00E-01 [C]	--	5.37E-05
Isophorone	26	8,450	3.68E-05	2.36E-02	4.10E-03 [C]	1.51E-07	9.69E-05
Methylene chloride	522	200,000	7.30E-04	5.59E-01	7.50E-03 [B2]	5.47E-06	4.18E-03
PCBs	1.9	17,000	2.66E-06	4.75E-02	7.70E+00 [B2]	2.04E-05	3.06E-01
1,1,2,2-Tetrachloroethane	17	7,350	2.38E-05	2.05E-02	2.00E-01 [C]	4.75E-06	4.10E-03
Tetrachloroethylene	16	24,500	2.26E-05	6.85E-02	5.10E-02 [B2]	1.15E-06	3.49E-03
Trichloroethylene	365	161,000	5.10E-04	4.50E-01	1.10E-02 [B2]	5.61E-06	4.94E-03
Vinyl chloride	106	7,290	1.48E-04	2.04E-02	2.30E+00 [A]	3.41E-04	4.58E-02
Total Excess Cancer Risk						6E-04	5E-01

CHEMICAL WITH NONCARCINOGENIC EFFECTS	WATER CONCENTRATION (ug/l)		CHRONIC DAILY INTAKE (mg/kg/day) (a)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Arsenic	31	3,100	3.00E-04	3.03E-02	1.00E-03	3.00E-01	3.03E+01
Bis(2-ethylhexyl)phthalate	17	654	1.67E-04	6.40E-03	2.00E-02	8.37E-03	3.20E-01
Butyl benzyl phthalate	NC	10	--	1.02E-04	2.00E-01	--	5.09E-04
Cadmium	3.5	16	3.42E-05	1.57E-04	5.00E-04	6.85E-02	3.13E-01
Chlorobenzene	9.8	4,020	9.59E-05	3.93E-02	2.00E-02	4.79E-03	1.97E+00
Chloroform	38	614,000	3.73E-04	6.01E+00	1.00E-02	3.73E-02	6.01E+02
2-Chloronaphthalene	NC	19	--	1.85E-04	NA	--	--
Chromium	26	450	2.57E-04	4.40E-03	5.00E-03	5.15E-02	8.81E-01
Cyanide	0.07	4.5	6.85E-07	4.40E-05	2.00E-02	3.42E-05	2.20E-03
1,2-Dichlorobenzene	35	192	3.41E-04	1.88E-03	9.00E-02	3.78E-03	2.09E-02
1,1-Dichloroethane	87	11,700	8.51E-04	1.14E-01	1.00E-01	8.51E-03	1.14E+00
1,1-Dichloroethylene	NC	32.0	--	3.13E-04	9.00E-03	--	3.48E-02
1,2-trans-Dichloroethylene	2,270	64,700	2.22E-02	6.33E-01	2.00E-02	1.11E+00	3.17E+01
Di-n-butyl phthalate	7.2	318	7.05E-05	3.11E-03	1.00E-01	7.05E-04	3.11E-02
2,4-Dimethylphenol	54	1,090	5.27E-04	1.07E-02	NA	--	--
Ethylbenzene	36	3,900	3.51E-04	3.82E-02	1.00E-01	3.51E-03	3.82E-01
Isophorone	26	8,450	2.57E-04	8.27E-02	2.00E-01	1.29E-03	4.13E-01
Lead	14	1,500	1.40E-04	1.47E-02	NA	--	--
Mercury	0.49	4.4	4.79E-06	4.31E-05	3.00E-04	1.60E-02	1.44E-01
Methylene chloride	522	200,000	5.11E-03	1.96E+00	6.00E-02	8.51E-02	3.26E+01
Methyl ethyl ketone	168	2,000,000	1.64E-03	1.96E+01	5.00E-02	3.29E-02	3.91E+02
Nickel	56	180	5.43E-04	1.76E-03	2.00E-02	2.72E-02	8.81E-02
Nitrobenzene	65	57,900	6.36E-04	5.67E-01	5.00E-04	1.27E+00	1.13E+03
Noncarcinogenic PAHs	31	2,707	3.00E-04	2.65E-02	4.00E-01	7.51E-04	6.62E-02
PCBs	1.9	17,000	1.86E-05	1.66E-01	1.00E-04	1.86E-01	1.66E+03
Phenol	510	17,100	4.99E-03	1.67E-01	6.00E-01	8.32E-03	2.79E-01
1,1,2,2-Tetrachloroethane	17	7,350	1.66E-04	7.19E-02	4.60E-04	3.62E-01	1.56E+02
Tetrachloroethylene	16	24,500	1.59E-04	2.40E-01	1.00E-02	1.59E-02	2.40E+01
Toluene	10,500	90,900	1.03E-01	8.89E-01	3.00E-01	3.42E-01	2.96E+00
1,1,1-Trichloroethane	59	81,200	5.75E-04	7.95E-01	9.00E-02	6.39E-03	8.83E+00
Trichloroethylene	365	161,000	3.57E-03	1.58E+00	7.35E-03	4.86E-01	2.14E+02
Xylenes	172	35,600	1.68E-03	3.48E-01	2.00E+00	8.42E-04	1.74E-01
Zinc	92	2,970	9.04E-04	2.91E-02	2.00E-01	4.52E-03	1.45E-01
Hazard Index						4E+00	4E+03

(a) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 days/yr).

NA = Not available.
NC = Not calculated because chemical was detected in only one sample.

methyl ethyl ketone and 1,1,1-trichloroethane), the reproductive system (due to PCBs) and the skin (due to arsenic). The average case hazard index values still exceeded one for the liver (due to trans-1,2-dichloroethylene and nitrobenzene), the blood (due to nitrobenzene), the central nervous system (due to trans-1,2-dichloroethylene) and the kidney (due to nitrobenzene).

Table 5-18 presents the intakes and risks assuming ground water is obtained from the till aquifer. In this case, the upper bound excess lifetime cancer risks are 4×10^{-5} and 4×10^{-3} for the average and RME cases, respectively. The risks for every chemical exceeded 1×10^{-6} for both the average and RME cases.

In addition, both the average case and RME case hazard index values exceeded one. When recalculated based on target organ categories, the values still exceeded one for liver effects (due to exposure to chloroform and trichloroethylene), indicating adverse effects might occur under these exposure scenarios.

Table 5-19 presents the chronic daily intakes and potential risks to a future worker via ingestion of ground water from the bedrock aquifer. For this exposure scenario, the upper bound excess lifetime cancer risks are 2×10^{-4} and 5×10^{-4}, for the average and RME cases, respectively. The risks exceeded 1×10^{-6} for every potential carcinogen detected in both of the bedrock samples. Of the chemicals detected in only one bedrock sample, only 1,1-dichloroethylene had an estimated risk greater than 1×10^{-6} associated with its single detected concentration. The hazard index values exceeded one for the average and RME

TABLE 5-18

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INGESTION
OF GROUND WATER BY FUTURE (ON-SITE) WORKERS
TILL AQUIFER

CHEMICAL WITH CARCINOGENIC EFFECTS	WATER CONCENTRATION (ug/1)		CHRONIC DAILY INTAKE (mg/kg/day) (a)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Chloroform	324	28,600	4.53E-04	8.00E-02	6.10E-03 [B2]	2.76E-06	4.88E-04
1,1-Dichloroethane	NC	27	--	7.55E-05	9.10E-02 [B2]	--	6.87E-06
1,2-Dichloroethane	144	9,230	2.01E-04	2.58E-02	9.10E-02 [B2]	1.83E-05	2.35E-03
1,1-Dichloroethylene	17	313	2.42E-05	8.75E-04	6.00E-01 [C]	1.45E-05	5.25E-04
Methylene chloride	101	1,210	1.41E-04	3.38E-03	7.50E-03 [B2]	1.06E-06	2.54E-05
PCBs	NC	1.8	--	5.03E-06	7.70E+00 [B2]	--	3.87E-05
Tetrachloroethylene	27	996	3.73E-05	2.78E-03	5.10E-02 [B2]	1.90E-06	1.42E-04
Trichloroethylene	410	16,400	5.73E-04	4.58E-02	1.10E-02 [B2]	6.30E-06	5.04E-04
Vinyl chloride	NC	54	--	1.52E-04	2.30E+00 [A]	--	3.49E-04
Total Excess Cancer Risk						4E-05	4E-03

CHEMICAL WITH NONCARCINOGENIC EFFECTS	WATER CONCENTRATION (ug/1)		CHRONIC DAILY INTAKE (mg/kg/day) (a)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Chlorobenzene	4.6	40	4.50E-05	3.88E-04	2.00E-02	2.25E-03	1.94E-02
Chloroform	324	28,600	3.17E-03	2.80E-01	1.00E-02	3.17E-01	2.80E+01
1,2-Dichlorobenzene	5.4	7.5	5.28E-05	7.30E-05	9.00E-02	5.87E-04	8.11E-04
1,1-Dichloroethane	NC	27	--	2.64E-04	1.00E-01	--	2.64E-03
1,1-Dichloroethylene	17	313	1.69E-04	3.06E-03	9.00E-03	1.88E-02	3.40E-01
1,2-trans-Dichloroethylene	12	190	1.14E-04	1.86E-03	2.00E-02	5.68E-03	9.30E-02
Methylene chloride	101	1,210	9.88E-04	1.18E-02	6.00E-02	1.65E-02	1.97E-01
Nitrobenzene	7.2	23	7.05E-05	2.28E-04	5.00E-04	1.41E-01	4.56E-01
PCBs	NC	1.8	--	1.76E-05	1.00E-04	--	1.76E-01
Phenol	NC	2.2	--	2.11E-05	6.00E-01	--	3.52E-05
Tetrachloroethylene	27	996	2.61E-04	9.75E-03	1.00E-02	2.61E-02	9.75E-01
Toluene	3.1	10	3.03E-05	9.88E-05	3.00E-01	1.01E-04	3.29E-04
1,1,1-Trichloroethane	30	417	2.89E-04	4.08E-03	9.00E-02	3.21E-03	4.53E-02
Trichloroethylene	410	16,400	4.01E-03	1.60E-01	7.35E-03	5.46E-01	2.18E+01
Zinc	30	57	2.89E-04	5.58E-04	2.00E-01	1.44E-03	2.79E-03
Hazard Index						1E+00	5E+01

(a) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 days/yr).

NC = Not calculated because chemical was detected in only one sample.

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TABLE 5-19

POTENTIAL EXPOSURES AND RISKS ASSOCIATED WITH INGESTION
OF GROUND WATER BY FUTURE (ON-SITE) WORKERS
BEDROCK AQUIFER

CHEMICAL WITH CARCINOGENIC EFFECTS	WATER CONCENTRATION (ug/l)		CHRONIC DAILY INTAKE (mg/kg/day) (a)		SLOPE FACTOR (mg/kg/day)-1 [Weight of Evidence]	EXCESS UPPER BOUND LIFETIME CANCER RISK	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Chloroform	670	830	9.37E-04	2.32E-03	6.10E-03 [B2]	5.71E-06	1.42E-05
1,2-Dichloroethane	420	460	5.87E-04	1.29E-03	9.10E-02 [B2]	5.34E-05	1.17E-04
1,1-Dichloroethylene	NC	2	--	5.59E-06	6.00E-01 [C]	--	3.35E-06
Methylene chloride	NC	21	--	5.87E-05	7.50E-03 [B2]	--	4.40E-07
Tetrachloroethylene	NC	2	--	5.59E-06	5.10E-02 [B2]	--	2.85E-07
Trichloroethylene	240	310	3.35E-04	8.67E-04	1.10E-02 [B2]	3.69E-06	9.53E-06
Vinyl chloride	28	56	3.91E-05	1.57E-04	2.30E+00 [A]	9.00E-05	3.60E-04
Total Excess Cancer Risk						2E-04	5E-04

CHEMICAL WITH NONCARCINOGENIC EFFECTS	WATER CONCENTRATION (ug/l)		CHRONIC DAILY INTAKE (mg/kg/day) (a)		REFERENCE DOSE (mg/kg/day)	HAZARD INDEX	
	Geometric Mean	Maximum	Average Case	Reasonable Maximum Case		Average Case	Reasonable Maximum Case
Chloroform	670	830	6.56E-03	8.12E-03	1.00E-02	6.56E-01	8.12E-01
Chromium	NC	28	--	2.70E-04	5.00E-03	--	5.40E-02
1,1-Dichloroethylene	NC	2	--	1.96E-05	9.00E-03	--	2.17E-03
1,2-trans-Dichloroethylene	NC	3	--	2.94E-05	2.00E-02	--	1.47E-03
Lead	NC	2.6	--	2.54E-05	NA	--	--
Methylene chloride	NC	21	--	2.05E-04	6.00E-02	--	3.42E-03
Tetrachloroethylene	NC	2	--	1.96E-05	1.00E-02	--	1.96E-03
Toluene	NC	15	--	1.47E-04	3.00E-01	--	4.89E-04
1,1,1-Trichloroethane	NC	8	--	7.83E-05	9.00E-02	--	8.70E-04
Trichloroethylene	240	310	2.35E-03	3.03E-03	7.35E-03	3.20E-01	4.13E-01
Zinc	NC	7.8	--	7.63E-05	2.00E-01	--	3.82E-04
Hazard Index						1E+00	1E+00

(a) Chronic daily intakes were averaged over a 70-year lifetime (25,550 days) for carcinogens and over the period of exposure for noncarcinogens (i.e., 10 or 20 yrs * 365 dys/yr).

NA = Not available.
NC = Not calculated because chemical was detected (or analyzed for, for inorganics) in only one sample.

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cases of worker ingestion of bedrock aquifer ground water for the liver (due to chloroform and trichloroethylene).

5.2.2.6 Potential Exposures Via Multiple Pathways

The risks presented in this report have, up to this point, been presented separately for each individual exposure pathway. It is possible, however, that a receptor could be exposed through more than a single pathway. For example, under current site use conditions, off-site receptors could potentially be exposed via inhalation of volatilized organics released from site soils as well as inhalation of suspended dusts. Tables 5-20 and 5-21 summarize these potential risks for off-site workers and off-site residents, respectively. As seen in these tables, the average case risk estimates and the RME case risk estimates, have been summed separately. The upper bound excess lifetime cancer risks for nearby off-site workers ranged from $3x10^{-7}$ to $5x10^{-6}$ for the average and RME cases, respectively. For nearby off-site residents, the upper bound excess lifetime cancer risks ranged from $1x10^{-7}$ to $8x10^{-7}$ for the average and RME cases, respectively. The chemicals that accounted for the major portion of the estimated risks were chromium, 1,1-dichloroethylene, and vinyl chloride. The hazard index values for nearby off-site workers and residents did not exceed one, indicating that adverse noncarcinogenic effects would be unlikely to occur for these two pathways.

Under future site use conditions, a future on-site worker could also be exposed through more than one pathway. Table 5-22 summarizes the potential

TABLE 5-20

POTENTIAL RISKS ASSOCIATED WITH EXPOSURES VIA
MULTIPLE PATHWAYS BY NEARBY (OFF-SITE) WORKERS

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	AVERAGE CASE EXCESS UPPER BOUND LIFETIME CANCER RISK			REASONABLE MAXIMUM CASE EXCESS UPPER BOUND LIFETIME CANCER RISK		
	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Cancer Risk	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Cancer Risk
Aldrin	6E-11	3E-11	9E-11	9E-10	5E-10	1E-09
Arsenic	--	4E-09	4E-09	--	6E-08	6E-08
Benzene	5E-10	--	5E-10	7E-09	--	7E-09
Bis(2-ethylhexyl)phthalate	--	--	--	--	--	--
Butyl benzyl phthalate	--	--	--	--	--	--
Cadmium	--	2E-09	2E-09	--	2E-08	2E-08
Carcinogenic PAHs	--	--	--	--	--	--
Chloroform	8E-09	--	8E-09	1E-07	--	1E-07
Chromium	--	1E-07	1E-07	--	2E-06	2E-06
1,2-Dichloroethane	1E-09	--	1E-09	2E-08	--	2E-08
1,1-Dichloroethylene	1E-07	--	1E-07	2E-06	--	2E-06
Dieldrin	2E-10	1E-10	3E-10	3E-09	2E-09	5E-09
Methylene chloride	8E-09	--	8E-09	1E-07	--	1E-07
Nickel	--	9E-10	9E-10	--	1E-08	1E-08
PCBs	--	--	--	--	--	--
1,1,2,2-Tetrachloroethane	6E-11	--	6E-11	9E-10	--	9E-10
Tetrachloroethylene	2E-10	--	2E-10	2E-09	--	2E-09
Trichloroethylene	9E-09	--	9E-09	1E-07	--	1E-07
Vinyl chloride	9E-08	--	9E-08	1E-06	--	1E-06
Total Excess Lifetime Cancer Risk			3E-07			5E-06

CHEMICAL WITH NONCARCINOGENIC EFFECTS	AVERAGE CASE HAZARD INDEX			REASONABLE MAXIMUM CASE HAZARD INDEX		
	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Hazard Index	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Hazard Index
Antimony	--	--	--	--	--	--
Bis(2-ethylhexyl)phthalate	--	--	--	--	--	--
Butyl benzyl phthalate	--	--	--	--	--	--
Chlorobenzene	1E-05	--	1E-05	8E-05	--	8E-05
2-Chloronaphthalene	--	--	--	--	--	--
Cyanide	--	--	--	--	--	--
1,2-Dichlorobenzene	6E-07	4E-09	6E-07	4E-06	3E-08	4E-06
1,1-Dichloroethane	1E-05	--	1E-05	8E-05	--	8E-05
1,2-trans-Dichloroethylene	--	--	--	--	--	--
Di-n-butyl phthalate	--	--	--	--	--	--
Di-n-octyl phthalate	--	--	--	--	--	--
2,4-Dimethylphenol	--	--	--	--	--	--
Ethylbenzene	--	--	--	--	--	--
Lead	--	--	--	--	--	--
Mercury	--	--	--	--	--	--
Methyl ethyl ketone	2E-06	--	2E-06	1E-05	--	1E-05
Methylene chloride	5E-06	--	5E-06	3E-05	--	3E-05
Noncarcinogenic PAHs	--	--	--	--	--	--
PCBs	--	--	--	--	--	--
Phenol	--	--	--	--	--	--
Selenium	--	1E-07	1E-07	--	1E-06	1E-06
Silver	--	--	--	--	--	--
Toluene	2E-06	--	2E-06	1E-05	--	1E-05
1,1,1-Trichloroethane	1E-06	--	1E-06	9E-06	--	9E-06
Xylenes	1E-05	--	1E-05	7E-05	--	7E-05
Zinc	--	--	--	--	--	--
Total Hazard Index			4E-05			3E-04

-- = Chemical not relevant for this exposure pathway (e.g., because chemical does not volatilize or would not remain adsorbed to suspended soil particles) or health criteria (i.e., slope factors or RfDs) not available.

TABLE 5-21

POTENTIAL RISKS ASSOCIATED WITH EXPOSURES VIA
MULTIPLE PATHWAYS BY NEARBY (OFF-SITE) RESIDENTS

CURRENT SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	AVERAGE CASE EXCESS UPPER BOUND LIFETIME CANCER RISK			REASONABLE MAXIMUM CASE EXCESS UPPER BOUND LIFETIME CANCER RISK		
	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Cancer Risk	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Cancer Risk
Aldrin	2E-11	9E-12	3E-11	1E-10	7E-11	2E-10
Arsenic	--	1E-09	1E-09	--	9E-09	9E-09
Benzene	1E-10	--	1E-10	1E-09	--	1E-09
Bis(2-ethylhexyl)phthalate	--	--	--	--	--	--
Butyl benzyl phthalate	--	--	--	--	--	--
Cadmium	--	5E-10	5E-10	--	4E-09	4E-09
Carcinogenic PAHs	--	--	--	--	--	--
Chloroform	2E-09	--	2E-09	2E-08	--	2E-08
Chromium	--	4E-08	4E-08	--	3E-07	3E-07
1,2-Dichloroethane	3E-10	--	3E-10	2E-09	--	2E-09
1,1-Dichloroethylene	3E-08	--	3E-08	2E-07	--	2E-07
Dieldrin	5E-11	3E-11	8E-11	4E-10	3E-10	7E-10
Methylene chloride	2E-09	--	2E-09	2E-08	--	2E-08
Nickel	--	3E-10	3E-10	--	2E-09	2E-09
PCBs	--	--	--	--	--	--
1,1,2,2-Tetrachloroethane	2E-11	--	2E-11	1E-10	--	1E-10
Tetrachloroethylene	5E-11	--	5E-11	4E-10	--	4E-10
Trichloroethylene	3E-09	--	3E-09	2E-08	--	2E-08
Vinyl chloride	3E-08	--	3E-08	2E-07	--	2E-07
Total Excess Lifetime Cancer Risk			1E-07			8E-07

CHEMICAL WITH NONCARCINOGENIC EFFECTS	AVERAGE CASE HAZARD INDEX			REASONABLE MAXIMUM CASE HAZARD INDEX		
	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Hazard Index	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Total Hazard Index
Antimony	--	--	--	--	--	--
Bis(2-ethylhexyl)phthalate	--	--	--	--	--	--
Butyl benzyl phthalate	--	--	--	--	--	--
Chlorobenzene	4E-06	--	4E-06	9E-06	--	9E-06
2-Chloronaphthalene	--	--	--	--	--	--
Cyanide	--	--	--	--	--	--
1,2-Dichlorobenzene	2E-07	1E-09	2E-07	5E-07	3E-09	5E-07
1,1-Dichloroethane	3E-06	--	3E-06	8E-06	--	8E-06
1,2-trans-Dichloroethylene	--	--	--	--	--	--
Di-n-butyl phthalate	--	--	--	--	--	--
Di-n-octyl phthalate	--	--	--	--	--	--
2,4-Dimethylphenol	--	--	--	--	--	--
Ethylbenzene	--	--	--	--	--	--
Lead	--	--	--	--	--	--
Mercury	--	--	--	--	--	--
Methyl ethyl ketone	5E-07	--	5E-07	1E-06	--	1E-06
Methylene chloride	1E-06	--	1E-06	3E-06	--	3E-06
Noncarcinogenic PAHs	--	--	--	--	--	--
PCBs	--	--	--	--	--	--
Phenol	--	--	--	--	--	--
Selenium	--	5E-08	5E-08	--	1E-07	1E-07
Silver	--	--	--	--	--	--
Toluene	5E-07	--	5E-07	1E-06	--	1E-06
1,1,1-Trichloroethane	4E-07	--	4E-07	1E-06	--	1E-06
Xylenes	3E-06	--	3E-06	7E-06	--	7E-06
Zinc	--	--	--	--	--	--
Total Hazard Index			1E-05			3E-05

-- = Chemical not relevant for this exposure pathway (e.g., because chemical does not volatilize or would not remain adsorbed to suspended soil particles) or health criteria (i.e., slope factors or RfDs) not available.

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TABLE 5-22

POTENTIAL RISKS ASSOCIATED WITH EXPOSURES VIA
MULTIPLE PATHWAYS BY FUTURE (ON-SITE) WORKERS

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH CARCINOGENIC EFFECTS	AVERAGE CASE EXCESS UPPER BOUND LIFETIME CANCER RISK					REASONABLE MAXIMUM CASE EXCESS UPPER BOUND LIFETIME CANCER RISK				
	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Direct Soil Contact	Groundwater Ingestion (Bedrock)	Total Cancer Risk	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Direct Soil Contact	Groundwater Ingestion (Bedrock)	Total Cancer Risk
Aldrin	9E-10	4E-10	2E-09	--	3E-09	1E-08	6E-09	3E-04	--	3E-04
Arsenic	--	5E-08	5E-08	--	1E-07	--	8E-07	3E-05	--	3E-05
Benzene	7E-09	--	8E-11	--	7E-09	1E-07	--	2E-06	--	2E-06
Bis(2-ethylhexyl)phthalate	--	--	8E-10	--	8E-10	--	--	2E-06	--	2E-06
Butyl benzyl phthalate	--	--	--	--	--	--	--	--	--	--
Cadmium	--	2E-08	--	--	2E-08	--	3E-07	--	--	3E-07
Carcinogenic PAHs	--	--	3E-07	--	3E-07	--	--	2E-04	--	2E-04
Chloroform	1E-07	--	8E-12	6E-06	6E-06	2E-06	--	1E-07	1E-05	1E-05
Chromium	--	2E-06	--	--	2E-06	--	3E-05	--	--	3E-05
1,1-Dichloroethane	--	--	2E-10	--	2E-10	--	--	7E-06	--	7E-06
1,2-Dichloroethane	1E-08	--	2E-10	5E-05	5E-05	2E-07	--	1E-06	1E-04	1E-04
1,1-Dichloroethylene	1E-06	--	2E-10	--	1E-06	2E-05	--	1E-07	3E-06	2E-05
Dieldrin	2E-09	2E-09	1E-08	--	1E-08	3E-08	2E-08	3E-04	--	3E-04
Isophorone	--	--	--	--	--	--	--	--	--	--
Methylene chloride	1E-07	--	3E-11	--	1E-07	2E-06	--	2E-08	4E-07	2E-06
Nickel	--	1E-08	--	--	1E-08	--	2E-07	--	--	2E-07
PCBs	--	--	2E-06	--	2E-06	--	--	9E-02	--	9E-02
1,1,2,2-Tetrachloroethane	8E-10	--	--	--	8E-10	1E-08	--	1E-07	--	1E-07
Tetrachloroethylene	2E-09	--	2E-09	--	4E-09	3E-08	--	3E-04	3E-07	3E-04
Trichloroethylene	1E-07	--	9E-11	4E-06	4E-06	2E-06	--	3E-05	1E-05	4E-05
Vinyl chloride	1E-06	--	--	9E-05	9E-05	2E-05	--	--	4E-04	4E-04
Total Excess Lifetime Cancer Risk					2E-04					9E-02

-- = Chemical not relevant for this exposure pathway (e.g., because chemical does not volatilize or would not remain adsorbed to suspended soil particles), health criteria (i.e., slope factors) not available, chemical was detected in only one sample (affects average case only), or chemical was not detected.

TABLE 5-22 (Continued)

POTENTIAL RISKS ASSOCIATED WITH EXPOSURES VIA
MULTIPLE PATHWAYS BY FUTURE (ON-SITE) WORKERS

FUTURE SITE AND LAND USE CONDITIONS

CHEMICAL WITH NONCARCINOGENIC EFFECTS	AVERAGE CASE HAZARD INDEX					REASONABLE MAXIMUM CASE HAZARD INDEX				
	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Direct Soil Contact	Groundwater Ingestion (Bedrock)	Total Hazard Index	Inhalation of Volatilized Organics	Inhalation of Suspended Soil	Direct Soil Contact	Groundwater Ingestion (Bedrock)	Total Hazard Index
Aldrin	--	--	3E-05	--	3E-05	--	--	2E+00	--	2E+00
Antimony	--	--	3E-04	--	3E-04	--	--	4E-02	--	4E-02
Arsenic	--	--	2E-04	--	2E-04	--	--	6E-02	--	6E-02
Bis(2-ethylhexyl)phthalate	--	--	2E-05	--	2E-05	--	--	2E-02	--	2E-02
Butyl benzyl phthalate	--	--	9E-07	--	9E-07	--	--	6E-04	--	6E-04
Cadmium	--	--	2E-04	--	2E-04	--	--	9E-02	--	9E-02
Chlorobenzene	2E-04	--	1E-06	--	2E-04	1E-03	--	7E-02	--	7E-02
Chloroform	--	--	9E-07	7E-01	7E-01	--	--	8E-03	8E-01	8E-01
2-Chloronaphthalene	--	--	--	--	--	--	--	--	--	--
Chromium	--	--	4E-04	--	4E-04	--	--	1E-01	5E-02	2E-01
Cyanide	--	--	3E-06	--	3E-06	--	--	2E-04	--	2E-04
1,2-Dichlorobenzene	8E-06	5E-08	1E-06	--	9E-06	6E-05	4E-07	2E-03	--	2E-03
1,1-Dichloroethane	1E-04	--	2E-07	--	1E-04	1E-03	--	3E-03	--	4E-03
1,1-Dichloroethylene	--	--	2E-07	--	2E-07	--	--	9E-05	2E-03	2E-03
1,2-trans-Dichloroethylene	--	--	1E-07	--	1E-07	--	--	5E-05	1E-03	1E-03
Dieldrin	--	--	9E-05	--	9E-05	--	--	1E+00	--	1E+00
Di-n-butyl phthalate	--	--	4E-06	--	4E-06	--	--	2E-03	--	2E-03
Di-n-octyl phthalate	--	--	--	--	--	--	--	--	--	--
2,4-Dimethylphenol	--	--	--	--	--	--	--	--	--	--
Ethylbenzene	--	--	8E-07	--	8E-07	--	--	3E-02	--	3E-02
Isophorone	--	--	--	--	--	--	--	--	--	--
Lead	--	--	--	--	--	--	--	--	--	--
Mercury	--	--	1E-03	--	1E-03	--	--	3E-01	--	3E-01
Methyl ethyl ketone	2E-05	--	4E-07	--	2E-05	2E-04	--	7E-04	--	9E-04
Methylene chloride	6E-05	--	5E-07	--	6E-05	4E-04	--	2E-04	3E-03	4E-03
Nickel	--	--	2E-05	--	2E-05	--	--	2E-03	--	2E-03
Nitrobenzene	--	--	--	--	--	--	--	2E-01	--	2E-01
Noncarcinogenic PAHs	--	--	2E-06	--	2E-06	--	--	6E-04	--	6E-04
PCBs	--	--	2E-02	--	2E-02	--	--	4E+02	--	4E+02
Phenol	--	--	2E-08	--	2E-08	--	--	2E-04	--	2E-04
Selenium	--	2E-06	4E-06	--	6E-06	--	1E-05	2E-03	--	2E-03
Silver	--	--	1E-05	--	1E-05	--	--	6E-03	--	6E-03
1,1,2,2-Tetrachloroethane	--	--	--	--	--	--	--	4E-03	--	4E-03
Tetrachloroethylene	--	--	2E-05	--	2E-05	--	--	2E+00	2E-03	2E+00
Toluene	2E-05	--	5E-07	--	2E-05	2E-04	--	5E-02	5E-04	5E-02
1,1,1-Trichloroethane	2E-05	--	--	--	2E-05	1E-04	--	6E-05	9E-04	1E-03
Trichloroethylene	--	--	8E-06	3E-01	3E-01	--	--	1E+00	4E-01	1E+00
Xylenes	1E-04	--	2E-07	--	1E-04	1E-03	--	7E-03	--	8E-03
Zinc	--	--	5E-05	4E-04	5E-04	--	--	2E-02	4E-04	2E-02
Total Hazard Index					1E+00					4E+02

-- = Chemical not relevant for this exposure pathway (e.g., because chemical does not volatilize or would not remain adsorbed to suspended soil particles), health criteria (i.e., RfDs) not available, chemical was detected in only one sample (affects average case only), or chemical was not detected.

risks for a possible future on-site worker exposed through four pathways: direct contact with surface soil; inhalation of volatilized organics; inhalation of suspended dusts; and ingestion of on-site ground water (assuming use of the bedrock aquifer). The upper bound excess lifetime cancer risks for all four pathways combined are 2×10^{-4} for the average case (due primarily to chloroform, chromium, 1,2-dichloroethane, 1,1-dichloroethylene, PCBs, trichloroethylene and vinyl chloride) and 9×10^{-2} for the RME case (the risks exceeded 10^{-6} for almost all of the chemicals). The most important pathway of exposure varied depending upon the specific chemical. The hazard index value for future on-site workers was equal to one for the average case primarily due to ingestion of chloroform and trichloroethylene in groundwater. The hazard index value for noncarcinogenic effects exceeded one for the RME case, primarily due to direct contact exposure to aldrin, antimony, lead, and tetrachloroethylene in soil and ingestion of chloroform and trichloroethylene in ground water. Assuming a future worker would use the till, rather than bedrock aquifer, the upper bound excess lifetime cancer risks for the four pathways combined would range from 5×10^{-5} to 1×10^{-1} for the average and RME cases, respectively. If the water table aquifer provided drinking water, the excess lifetime cancer risks for the four pathways combined would range from 6×10^{-4} to 6×10^{-1} for the average and RME cases, respectively. The hazard index values for both the average and RME cases and both aquifers exceeded one, indicating that adverse effects could occur under the exposure conditions evaluated.

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6.0 ECOLOGICAL RISK ASSESSMENT

The steps followed in an ecological risk assessment roughly parallel those for a human health risk assessment, in that information on exposure and toxicity are combined to generate an estimate of potential impacts. Ecological risks are usually evaluated separately because of the potential diversity of the receptors. Another major difference between an ecological risk assessment and a human health risk assessment is the receptor type focused on. In a human health risk assessment, potential exposures to both individuals and populations may be estimated. While protection of individual environmental receptors also may be important (e.g., the death of one individual of an endangered species), in most cases, an ecological risk assessment focuses at the population and ecosystem levels. Because there is a paucity of toxicity data relevant to wildlife and it is difficult to draw inferences at the population and ecosystem levels, this ecological risk assessment will be largely qualitative.

6.1 SELECTION OF CHEMICALS OF CONCERN

For the selection of chemicals of concern for the ecological risk assessment, the list of chemicals selected for the human health risk assessment was used as a starting point. Then, chemicals which were not selected (for example, copper because it is an essential human nutrient and unlikely to cause adverse human health effects at the levels observed on site) were considered for addition to the list for the ecological risk assessment because of their

toxicity to aquatic life. Thus, copper which was detected in surface water and sediments is added to the list of chemicals of concern for the ecological assessment. The available information concerning toxicity of chemicals to wildlife other than fish (particularly terrestrial wildlife) is quite limited. Therefore, the ecological risk assessment will necessarily be limited to those chemicals for which toxicity information is available or which can easily be inferred by interspecies extrapolation.

6.2 ENVIRONMENTAL RECEPTOR ANALYSIS

The flora and fauna existing around the SCP site were inventoried most recently by biologists from the New Jersey Marine Science Consortium (1985).

It is not known whether or not endangered species occur in the immediate site vicinity. The peregrine falcon (Falco peregrinus), a federal listed endangered species (U.S. Fish and Wildlife Service 1989) has been frequently sighted in Bergen County (R. Kane, New Jersey Audubon Society, personal communication, February 14, 1989). Two pairs are known to be within flying distance of the site and one of these apparently frequents areas of the Hackensack Meadowlands within several miles of the site. Based on information from the New Jersey Department of Environmental Protection (NJDEP 1987, NJDEP no date), some areas of Bergen County are designated as breeding range for several species classified as State threatened species. A threatened species is one that becomes endangered if conditions surrounding the species begin to or continue to deteriorate. These species are: the savannah sparrow

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(<u>Passerculus</u> <u>sandwichensis</u>), the short-billed marsh wren (<u>Cistothorus</u> <u>platensis</u>), and the pied-billed grebe (<u>Podilymbus</u> <u>podiceps</u>).

The savannah sparrow prefers extensive short grass fields or relatively dry short grass salt marshes. A small area located in southern Bergen County is designated as a known breeding range for this bird. The short-billed marsh wren prefers drier portions of brackish marshes and wet inland meadows. Its nesting areas are usually associated with sedges and grasses. The pied-billed grebe prefers freshwater ponds, creeks, and marshy areas, especially in the Hackensack Meadowlands area of Bergen County. The breeding population of this species is classified by the State as endangered. There is only one known nesting population in the Meadowlands area, in Kearny Marsh located approximately 3 miles south of Berry's Creek, near the Bergen and Hudson County line. These species are considered threatened due to the increasing natural habitat loss as a result of human activities.

Animals considered in the ecological assessment are terrestrial wildlife species such as muskrat, fox, cottontail rabbit, field mice, other terrestrial mammals, songbirds, birds of prey, and aquatic wildlife such as fish and aquatic invertebrates. The primary type of vegetation in the area are reed grasses; the occurrence of trees is for the most part limited to areas along creekbeds.

The SCP site is located in what is classified as a Class IV wetlands (reed marsh), in the Hackensack Meadowlands area. This is an extensive area of

brackish water marshes that are drained by the Hackensack River and its tributaries. Two swamps, Walden Swamp and Eight Day Swamp, are located nearby. Surveys of aquatic life are currently not available for the immediate site area but a wide variety of aquatic life in water bodies near the site may exist. Berry's Creek, into which Peach Island Creek flows, contains an abundant population of killifish (Fundulus sp.), which are caught for use as bait for sport fishing. Carp (Cyprinus carpio) and white perch (Morone americana) are occasionally observed. Snapping turtles commonly occur in the tidal marsh habitat, and are likely to occur near Berry's Creek and Peach Island Creek. No macroscopic invertebrates have been observed in Berry's Creek. Snails (Physa spp.) have been observed near the nearby Sports Complex. Adult and larval mosquitos are present in large numbers over the entire Meadowlands region.

6.3 IDENTIFICATION OF ECOLOGICAL EXPOSURE PATHWAYS

The SCP site is bounded by Peach Island Creek on the northeast and enclosed by a fence on the other sides. The fence may deter some larger species of terrestrial wildlife (e.g. rabbit, fox) from entering the site, under present site and land use conditions. However, smaller species such as field mice, could enter through the fence and birds also have unrestricted access to the site. The area of the site which is adjacent to the Peach Island Creek is unfenced. Therefore, the potential for direct contact with chemicals on site exists. Under the no-action alternative, it will be assumed that the fence

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will not be maintained and thus access to the site will be unrestricted and the potential for direct exposures to wildlife may increase.

There are a number of direct and indirect pathways through which wildlife may be exposed to chemicals present at the SCP site. Direct pathways include direct contact with or ingestion of contaminated media such as soil, sediment, water, or air. Indirect pathways include those in which an animal consumes other previously contaminated organisms.

Exposure media and routes may differ between various organisms due to their physiological and behavioral differences. For example, fish may be exposed to contaminants either through the water column, from ingestion of other previously contaminated organisms, or from ingestion of sediments while feeding. Likewise, some predatory species may rely on both animals (i.e., rodents, other small mammals, insects, crustaceans, and snails) and plants for some portion of their dietary intake. However, the percentage of animal food versus plant food in the diet differs from species to species. Variables such as these must be considered in assessing exposure to ecological receptors.

6.3.1 Ground Water

The shallow aquifer at the SCP site extends to within one to two feet of the surface in some areas of the site. There is, therefore, the potential for exposure of terrestrial plants, and possible burrowing animals, to contaminated shallow ground water. However, as noted above, much of the area

of the site is currently unvegetated, and is unlikely to provide a desirable

habitat for significant populations of terrestrial animals. At present,

limited data are available regarding off-site ground water quality.

Therefore, given the available data, it is not possible to quantitatively

assess the potential impacts of ground water on environmental receptors at or

near the SCP site, except through the impacts of ground water drainage to

surface water at the site, as is discussed in Section 6.3.3.

6.3.2 <u>Soil</u>

It is presumed that no terrestrial animals live on site; none were observed

during the June 1988 site visit. The site is sparsely vegetated primarily

with grasses and weeds, which do not provide adequate cover for most

terrestrial animals. Should any terrestrial animals venture onto the site,

they may be directly exposed to contaminants in the soil. Direct contact with

contaminated soil and incidental ingestion could occur among dustbathing

animals, such as many bird species. Indirect exposure of animals to

contaminants in soil may occur via ingestion of grasses and other land plants

which may have bioaccumulated contaminants. Incidental ingestion of soil is a

possible exposure route for fastidious animals such as raccoons who may ingest

soil while grooming, for herbivorous animals such as rabbits who may ingest

soil while feeding on plants, or for seed-eating bird species who may ingest

soil while foraging for seeds on the ground. As with other exposure routes,

the importance of this exposure route varies between species because of

behavioral differences; populations of animals such as rabbits, which are both

herbivorous and frequent groomers, may be more greatly affected by

contaminated soil than other populations which contact soil less often.

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However, because the site is sparsely vegetated, has been disturbed by human activity, and no terrestrial mammals were observed, this assessment will not evaluate on-site soils as a source of contaminants for terrestrial animals.

Plants may be directly exposed to contaminants in soil via uptake through the roots. Chemicals may accumulate in various portions of different species of plants. Because phytotoxicity data are limited, and plant uptake values vary greatly from species to species, exposures via this pathway are difficult to quantify. At the site, the man-made fill material makes up a portion of the surface material, and as such, the surface of the site is not favorable for plant growth as evidenced by the sparse plant growth, consisting largely of common grasses and weeds. In order for other natural vegetation to become established, soil conditions on site would have to be at least partially restored. The extent to which sparse plant growth is a result of site contamination could not be determined.

6.3.3 Surface Water And Sediments

Fish and other aquatic organisms may be exposed to contaminants in surface water and sediments. Additionally, other environmental receptors are dependent on surface water as a source of drinking water. Terrestrial animals that rely on Peach Island Creek for drinking water may be exposed via ingestion to chemicals present in surface water. Among birds, intake from ingestion of contaminated water could be estimated, but this pathway is not believed to contribute significantly to overall intake relative to intake via

food for most chemicals. Risks to terrestrial animals, such as the muskrat, from drinking water from Peach Island Creek will be estimated.

Many chemicals in water are known to bioconcentrate directly in invertebrate organisms and in fish tissue via uptake through the gill membranes. Fish which inhabit Peach Island Creek are therefore exposed to chemicals present in surface water. Omnivorous mammals such as raccoons, or bird species such as ducks that consume previously contaminated aquatic organisms, would be exposed because of accumulation through the food chain. Likewise plants may also bioaccumulate chemicals via uptake of surface water or shallow ground water through the roots and translocation to various portions of the plant. The chemicals may then be ingested by animals higher up in the food chain.

Exposure to chemicals present in shallow sediments is another possible exposure pathway for ecological receptors. Deeper sediments (>6 inches deep) are generally less bioavailable and will not be evaluated for ecological risk, although it is possible that behavior (such as burrowing) and physical processes (such as storms) may result in exposure to chemicals in deeper sediments. In addition to benthic (bottom dwelling) aquatic organisms that are exposed to contaminants in the sediment, wading birds such as the great blue heron or other animals may also be exposed to contaminants from direct contact with sediment while foraging for food. However, data are inadequate to assess uptake in birds from this route.

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USEPA is in the process of developing sediment quality criteria (SQC) for the protection of aquatic life exposed to contaminated sediment. Interim SQCs have been developed for nonpolar, hydrophobic organics by the Equilibrium Partitioning Approach (USEPA 1988a). This approach assumes that (1) the toxicity and accumulation of contaminants by benthic organisms is correlated with the interstitial water concentration and (2) interstitial water concentrations are controlled by partitioning between sediment and water. The fraction of organic carbon (f_{oc}) in the sediment must be known or estimated for this approach. For this assessment a value of 10% will be used (i.e., f_{oc} = 0.1). This value is based on the qualitative observation that sediments were "waterlogged organic silt" (Dames and Moore 1990) and is the maximum value evaluated by USEPA (1988a) for the Equilibrium Partitioning Approach

The interim sediment quality criteria (SQC) are estimated by the following equation:

$$SQC = K_{oc} * C_w * f_{oc}$$

where

SQC   =   normalized Sediment Quality Criteria (mg/kg),

K_{oc} =   organic carbon partition coefficient (liter/kg),

C_w =   interstitial water concentration (mg/liter) which is expected to be protective of aquatic life (e.g. the AWQC), and

f_{oc} =   fraction organic carbon.

These SQC, normalized for the fraction organic carbon, are compared with the sediment concentrations in the same way that AWQC are compared with surface

water concentrations to estimate potential hazards. SQC for metals are currently being developed by USEPA. In the absence of any approved approach, the hazards of sediment metal concentrations will be discussed in relation to invertebrate sediment bioassay data.

6.4 ESTIMATION OF EXPOSURE POINT CONCENTRATIONS

Surface water, sediment, and soil are all potential media for exposure. Since the site is sparsely vegetated, has been disturbed by human activity, and no terrestrial mammals were observed, soils will not be considered to be a medium for exposure. Exposure point concentrations for aquatic life are the surface water and sediment concentrations presented in Tables 6-1 and 2-11, respectively.

As mentioned earlier, ducks, birds, and other animals may be exposed via ingestion of previously contaminated organisms. In consideration of the State endangered pied-billed grebe, exposure via this pathway will be estimated using surface water concentrations in Table 6-1 and applying available invertebrate bioconcentration factors to estimate the concentration within the aquatic organisms, and then estimating the total daily dose and comparing this with available toxicity information.

Exposure to muskrats through the drinking of surface water containing contaminants will also be estimated using the surface water data in Table 6-1 and parameters described in Section 6.6.

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TABLE 6-1

COMPARISON OF MAXIMUM SURFACE WATER CONCENTRATIONS
WITH CRITICAL TOXICITY VALUES

| | Surface Water Concentrations (ug/liter) | | | Ambient Water Quality Criteria (ug/l) | | | |
| | | | | Marine Waters | | Fresh Water | |
	Upstream	Near Site (a)	Confluence w/ Berry's Creek	Chronic	Acute	Chronic	Acute
Chlorobenzene	ND	12.2	ND	129 (b,e)	160 (b,e)	50 (b,e)	250 (b,e)
Chloroform	ND	3.58	ND	NA	NA	1,240 (b)	28,900 (b)
1,2,-trans-Dichloroethylene	ND	35.2	3.91	NA	224,000 (b,f)	NA	11,600 (b,f)
Methyl ethyl ketone	75	49.2	ND	NA	NA	NA	NA
Methylene chloride	4.6	12.9	14.9	NA	NA	NA	NA
Toluene	ND	48.1	ND	5,000 (b)	6,300 (b)	NA	17,500 (b)
1,1,1-Trichloroethane	ND	6.32	ND	NA	31,200 (b)	NA	18,000 (b,g)
Trichloroethylene	ND	3.83	ND	NA	2,000 (b)	21,900 (b)	45,000 (b)
m-Xylene	ND	10.7	ND	NA	NA	NA	NA
o+p-Xylenes	ND	10	ND	NA	NA	NA	NA
Chromium III	56 (d)	ND	28 (d)	NA	10,300 (b)	370 (c)	3,100 (c)
Chromium VI	56 (d)	ND	28 (d)	50	1,100	11	16
Copper	100	29	27	2.9	2.9	21 (c)	34 (c)
Mercury	4.8	0.96	1.1	0.025	2.1	0.012	2.4
Nickel	57	0.33	27	8.3	75	280 (c)	2,500 (c)
Zinc	370	160	150	86	95	190 (c)	210 (c)

Source: Quality Criteria for Water, Office of Water Regulations and Standards. 1986. EPA 440/5-86-001

(a) Maximum of measurements adjacent to site and 100 feet downstream.
(b) Lowest observed effect level.
(c) Value depends on water hardness; value given is for CaCO3 = 200 ug/l
(d) Value for total chromium.
(e) Value is for chlorinated benzenes.
(f) Value is for dichloroethenes.
(g) Value is for trichlorinated ethanes.

6.5 ENVIRONMENTAL HAZARD IDENTIFICATION

In this section, a brief description of the toxic effects of the site-related chemicals to non-human receptors are provided. In addition, any available chemical-specific standards, criteria and guidance will be identified. Ambient Water Quality Criteria for the protection of freshwater aquatic life have been established under the U.S. Clean Water Act for a number of chemicals. These criteria are developed to be protective of 95% of all aquatic species. Therefore, not only are fish protected, but aquatic invertebrates and plants are protected as well. These criteria may be compared with average and maximum surface water concentrations in the risk characterization section to determine the likelihood of adverse effects to aquatic life.

The USEPA (1988a) Equilibrium Partitioning Approach for deriving sediment quality criteria will be used. This approach is appropriate for nonpolar, hydrophobic organics; in this assessment a water solubility less than 100 ug/l was used as an operational definition of hydrophobicity. These criteria will be compared with average and maximum sediment concentrations in order to determine the likelihood of adverse effects to aquatic life.

No criteria have been developed specifically for the protection of terrestrial wildlife. Primary and secondary literature are usually the source of most of the relevant toxicity data for these receptors. For purposes of assessment, toxicity values are obtained from studies reporting no-observed-effect levels

(NOELs), lowest-observed-effect levels (LOELs), or median lethal doses ($LD_{50}s$) for terrestrial or avian species and are presented in the hazard identification section. The NOEL represents the highest dietary concentration or dose of a chemical not associated with an adverse effect in an animal, while the LOEL represents the lowest dietary concentration or dose reported to cause adverse effects. The LD_{50} represents the dose which was lethal to 50% of an experimental population.

The following guidelines are used to select critical toxicity values for use in the risk assessment. Chronic values are chosen over acute toxicity values when available. For those compounds for which only acute lethality values are available, the lowest LD_{50} value will be used and toxicity values will be derived by dividing the LD_{50} by an uncertainty factor. In evaluating the potential effects of chemicals on terrestrial wildlife, this risk assessment adopts the USEPA assumption that there is no acute hazard if the estimated dose is less than one-fifth of the LD_{50} for nonendangered species and less than one-tenth for endangered species (Urban and Cook 1986). Therefore, acute toxicity values are derived by dividing the LD_{50} by 5. If a chronic NOEL and a chronic LOEL are available, the NOEL is chosen as the critical toxicity value. The NOEL selected is the highest NOEL reported, and the LOEL selected is the lowest LOEL reported. A safety factor of 10 is applied when toxicity values are derived from subchronic rather than chronic toxicity data. A safety factor of 10 is also applied if data describe a lowest observed effect level (LOEL) rather than a NOEL. These safety factors are similar to those applied in the derivation of human health criteria.

For terrestrial wildlife, the toxicity assessment will focus on effects on mammals and birds since there are generally few data on the effects of contaminants on reptiles and amphibians. Toxicity values will be based on chronic studies in terrestrial mammalian wildlife whenever possible. Since there are few data for the chronic effects of contaminants on wild species of mammals, laboratory rodent studies will often be used as the basis for the toxicity values. Studies used by the USEPA in determining chronic oral reference doses (RfDs) will be used as the bases for the toxicity values, when such data exist. In general, these studies will provide NOELs from chronic studies with laboratory rodents. Safety factors of 10 will be applied when such data are LOELs rather than NOELs and when subchronic rather than chronic studies are used. Risks in Section 6.6 are estimated by comparing toxicity values with estimated dietary concentrations or doses in birds and mammals.

Ecological toxicity data are summarized in paragraphs similar to those used in the human hazard assessment. They contain a discussion of the known toxicological properties of the chemicals of concern, and present the available criteria and toxicity values. These values will be compared with estimated intake levels using various assumptions to determine the magnitude of ecological risk presented by the chemicals of concern. Because of the paucity of toxicity data, the chemicals of concern are grouped into broader categories of related chemicals. Where available, chemical-specific toxicity characteristics will be presented. This is possible for most of the inorganic chemicals, as they have been studied more extensively. Other chemical groups

which will be discussed are volatile organic chemicals, PAHs, and pesticides/PCBs. Paragraphs for these categories of the chemicals of concern at the SCP site are presented below.

6.5.1 Inorganic Chemicals

6.5.1.1 Arsenic

Arsenic at sufficiently high levels is toxic to both aquatic and terrestrial animal species, and induces its toxic effects via enzyme inhibition. It is generally agreed that inorganic arsenic is more toxic than organic arsenic, and trivalent arsenic (As (III)) is more toxic than pentavalent arsenic (As(V)). For trivalent arsenic, USEPA (1985a) has established a freshwater four-day average criterion of 190 ug/liter and a 1-hour average concentration criterion of 360 ug/liter. Data are insufficient to develop freshwater ambient water quality criteria for pentavalent arsenic, however USEPA (1986) reports freshwater acute and chronic LOELs of 850 ug/liter and 48 ug/liter, respectively. For As (III) in marine waters, USEPA (1986) developed a four-day average criterion of 36 ug/liter and a one-hour average criterion of 96 ug/liter. For As (V) in marine waters, USEPA (1986) reported acute and chronic LOELs of 2,319 ug/liter and 13 ug/liter, respectively.

Arsenic can bioaccumulate in aquatic vertebrates and invertebrates, but bioconcentration occurs to a greater extent in invertebrates. Bioconcentration factors (BCF) for As (III) of between 3 and 17 have been

reported for snails (USEPA 1984a). A maximum BCF of 7 is reported for As (V),
and a maximum BCF of 9 is reported for organoarsenicals (USEPA 1985a).

Quantitative data on the toxicity of arsenic to terrestrial and avian species
are limited. Most studies report median lethal concentrations or doses (LC_{50}s
and LD_{50}s respectively). Eisler (1988) reports that episodes of terrestrial
wildlife poisoning by arsenic are infrequent. Arsenic has been shown to
induce death in wild rabbits and hares following acute oral exposures; LD_{50}s
have been reported in the range of 10.5 to 40.4 mg/kg body weight (NRC 1977).

Lethal oral doses for most domestic mammals appear to be between 1 to 25 mg of
sodium arsenite per kg of body weight and 3 to 10 times that range as arsenic
trioxide. Some animals, particularly cattle, develop a preference for weeds
and vegetation sprayed with arsenic weed killer, most likely as a result of
the saltiness of the arsenic compounds (Selby et al. 1977). Eisler (1988)
recommends that straight feedstuffs fed to domestic livestock should contain
<2 mg total As/kg FW (fresh weight), and meals from grass, dried lucerne, or
dried clover should contain <4 mg total As/kg FW.

Median lethal concentrations (LC_{50}) in the diets of wild birds have been
reported in the range of 480 mg/kg for the bobwhite quail to 5,000 mg/kg for
mallards (Heath et al. 1972, Hill et al. 1975). Chronic poisonings in animals
have not been clearly documented. In mammals, effects such as weakness,
paralysis, conjunctivitis, dermatitis, decreased growth, and liver damage have
been correlated with chronic exposure to inorganic arsenicals (NRCC 1978).

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Arsenic bioaccumulates in terrestrial species but the available data indicate that biomagnification in the food chain does not occur. In fact, Andren et al. (1973) reported declining concentrations of arsenic in animals with higher trophic levels. Western grasshoppers poisoned by arsenic trioxide were fed to nestling northern bobwhites, mockingbirds, American robins, and other songbirds with essentially no deleterious effects (NRC 1977).

6.5.1.2 Beryllium

No freshwater or marine AWQC have been developed for beryllium because adequate data are not available. However, USEPA (1986) reported freshwater acute and chronic LOELs of 130 ug/liter and 5.3 ug/liter, respectively. Some of the data indicate that beryllium toxicity is inversely related to water hardness, but this relationship has not been quantitatively defined. In acute lethality tests with bluegill sunfish (Lepomis macrochirus) in hard water (400 mg/liter as $CaCO_3$) and soft water (20 mg/liter), 96-hour LC_{50} values of 12,000 and 1,300 ug/liter, respectively, were reported (USEPA 1980b). No chronic toxicity data were available in the literature reviewed.

6.5.1.3 Cadmium

Cadmium is a cumulative poison to aquatic organisms because excretion of this metal is virtually nonexistent. Studies reporting cadmium toxicity to aquatic organisms demonstrate an inverse relationship between cadmium toxicity and

water hardness. USEPA (1985b) recommended a 4-day average concentration criterion for cadmium not to exceed the value given by $e^{(0.7852[(\ln \text{ hardness})]-3.490)}$ and a 1-hour average concentration criterion not to exceed the value given by $e^{(1.128[(\ln \text{ hardness})]-3.828)}$ more than once every 3 years. Using an average water hardness of 200 mg/liter, the corresponding four-day and one-hour criteria are 2.0 and 8.6 ug/liter, respectively. In marine waters, four-day and one-hour criteria are 9.3 ug/liter and 43 ug/liter, respectively.

Cadmium can cause death and a variety of sublethal effects in birds exposed via the diet. Sublethal effects include growth retardation, anemia, and testicular damage. Several studies are available on the dietary toxicity of cadmium to ducks. In mallard ducklings fed dietary cadmium from day 1 of age for 12 weeks, hematological effects were observed at 8 weeks and mild to severe kidney lesions developed at 12 weeks (Cain et al. 1983). However, no hematological effects were observed in adult mallards exposed to 200 ppm in the diet for 90 days (White and Finley 1978). In other duck species, no effects on growth or kidneys were observed in wood ducks (Aix sponsa) exposed from age 1 week to age 7 weeks to 1, 10, or 100 ppm cadmium in the diet (Mayack et al. 1981). Altered avoidance behavior in the form of hyperresponsiveness was observed in young black ducks (Anas rubripes) born to parents exposed to 4 ppm cadmium in the diet for 4 months prior to birth of their young (Heinz and Haseltine 1983).

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No data were located on cadmium toxicity in raptor species, such as falcons and eagles, nor were effects identified in other bird species which occurred at dietary concentrations lower than those observed to cause toxic effects in ducks. The LOEL in ducks was 4 ppm in the diet. This level resulted in altered avoidance behavior in young black ducks.

6.5.1.4 Chromium

For freshwater aquatic organisms, hexavalent chromium (Cr VI) appears to be more toxic than trivalent chromium (Cr III). Impaired growth, increased locomotor activity, and enzyme inhibition have been reported in fish following chronic exposure to chromium. USEPA (1985c) has established a freshwater four-day criterion for Cr (VI) of 11 ug/liter and a 1-hour average criterion of 16 ug/liter not to be exceeded more than once every three years. For Cr (III) in freshwater at a hardness of 200 mg/l $CaCO_3$, the four-day criterion is 370 ug/liter and the one-hour criterion is 3,100 ug/liter. For Cr (VI) marine waters, USEPA (1985c) developed a four-day average criterion of 50 ug/liter and a one-hour average criterion of 1,100 ug/liter. For Cr (III), USEPA (1985c) reported an acute LOEL of 10,300 ug/liter.

Few data are available on the toxic effects of chromium in birds. Adult black ducks fed diets containing 10 or 50 ppm chromium (III) for five months were normal in survival, reproduction, and blood chemistry (Haseltine et al. 1985). However, ducklings from treated groups that were fed chromium at the original parental doses experienced altered growth and reduced survival. No effect on

avoidance response was observed in young black ducklings fed chromium at 20 or 100 ppm in the diet for seven days that were born to parents fed these same doses (Heinz and Haseltine 1981). No data on the toxicity of chromium to raptor species were located. The 10 ppm dietary LOEL in ducks was the lowest LOEL for birds indicated in the available literature.

A chronic oral mammalian toxicity value of 0.24 mg/kg/day was derived based on information from the IRIS database (USEPA 1988b). A NOEL of 0.24 mg/kg/day from a chronic rat study was the basis for the toxicity value.

6.5.1.5 Copper

The primary mechanism of copper toxicity in aquatic organisms is osmoregulatory dysfunction and failure (Leland and Kuwabara 1985). Continued ingestion of copper in excess of nutritional requirements leads to accumulation, especially in the liver (Leland and Kuwabara 1985). Copper does not appear to bioconcentrate directly from water. Copper toxicity decreases with increasing water hardness. USEPA (1985d) recommended that the 4-day average concentration of copper (in ug/liter) should not exceed the value given by $e^{(0.8545[\ln(\text{hardness})]-1.465)}$, and the 1-hour average concentration should not exceed the value given by $e^{(0.942[\ln(\text{hardness})]-1.464)}$. At a water hardness of 200 mg/liter, the corresponding values are 21 and 34 ug/liter respectively. For marine waters USEPA (1985d) reported a criterion of 2.9 ug/liter both as a four-day and one-hour average concentration.

NAS (1980) reviewed the toxic effects of copper in livestock and poultry. Growth rates of young ducks increased when they received 100 ppm oral copper (given as cupric sulfate) for 8 weeks (King 1975). Mayo et al. (1956) reported that 324 ppm in the diet caused growth retardation and muscular dystrophy in growing chickens. NAS (1980) recommended 300 ppm as a maximum tolerable dietary level for poultry. Since no data were found on the effects of copper on raptors, the value of 300 ppm will be used as a toxicity value for this risk assessment.

A chronic oral toxicity value of 6 mg/kg/day for mammalian wildlife is derived by multiplying the NAS (1980) maximum tolerable level for rabbits of 200 ppm by a dietary conversion factor of 0.03 mg/kg body weight per ppm (Lehman 1954).

6.5.1.6 Lead

The primary mechanism of acute toxicity of lead to freshwater organisms is unknown. Lead toxicity decreases with increasing water hardness. USEPA (1986) has established four-day and one-hour average concentration criteria for lead not to be exceeded by the values given by $e^{(1.266[\ln(hardness)]-4.661)}$ and $e^{(1.266[\ln(hardness)]-1.416)}$, respectively, more than once every three years. Using an average water hardness of 200 mg/liter, the 4-day and 1-hour criteria correspond to 7.7 and 200 ug/liter, respectively. For marine waters, USEPA (1986) has established four-day and one-hour average criteria of 5.6 ug/liter and 140 ug/liter, respectively.

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The majority of information on lead toxicity in birds is on body burdens in waterfowl that have ingested spent lead shot and died. However, limited dose-response information is available for a few species. Neurological effects were observed within 24 hours of dosing in mallard ducks that had ingested and absorbed lead shot for a total intake of 423.8 mg/kg bw (Mautino and Bell 1987). These effects decreased 8 days after dosing. Inhibition of delta-ALAD was apparent 1 week after dosing. Assuming a mallard weighs approximately 1.2 kg (calculated from Terres 1980) and consumes an amount of food equivalent to 10% of its body weight each day, the dosage of 423.8 mg/kg is equivalent to an approximate lead concentration in the food of 4,200 ppm. In American kestrels (Falco sparverius) fed 10 or 50 ppm lead in the diet for 7 months, no effects were noted with respect to survival, egg laying, initiation of incubation, or egg shell thickness (Pattee 1984). In 1-day-old American kestrels fed 125 or 625 mg/kg bw lead for 10 days, growth was seriously depressed by day 6, and hematocrit values were significantly depressed by day 10 (Hoffman et al. 1985). Forty percent of the birds receiving 625 mg/kg lead died within 6 days. No effects were observed in kestrels exposed to 25 mg/kg bw. Assuming complete absorption of the administered dose, and that a kestrel weighs 0.11 kg (calculated from Terres 1980) and consumes an amount of food equivalent to 10% of its body weight, the 25 mg/kg bw dosage corresponds to an approximate lead concentration in food of 280 ppm. Therefore, the 50 ppm level identified in the Pattee (1984) study is the highest NOEL identified for birds based on the studies reviewed.

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6.5.1.7 <u>Mercury</u>

Mercury is acutely and chronically toxic to freshwater fish. Signs of acute mercury poisoning in fish include flaring of gill covers, increased frequency of respiratory movements, loss of equilibrium, and sluggishness. Chronic poisoning can cause emaciation, brain lesions, cataracts, inability to capture food, and abnormal motor coordination. USEPA (1985e) has established four-day and one-hour concentration criteria of 0.012 and 2.4 ug/liter in freshwater. For marine waters, the four-day and one-hour criteria are 0.025 and 2.1 ug/liter, respectively.

Most of the data on the toxicity of mercury to birds is on the effects of methylmercury, an organic form which is much more toxic to birds and mammals than inorganic mercury. In toxicity studies with <u>Coturnix</u> (a type of quail), violent neurological dysfunction was observed beginning at 2 and ending at 4 hours following oral treatment with inorganic mercury (Eisler 1987a). An LD_{50} of 26 to 54 mg/kg bw was reported in this species (Eisler 1987a). No death occurred in this species when exposed to 32 ppm mercury in the diet from hatching for 9 weeks. In Japanese quail, an LD50 of 31.1 mg/kg bw was reported (Eisler 1987a). No other appropriate data were located on the toxicity of inorganic mercury to birds. The 32 ppm NOEL reported for <u>Coturnix</u> was the only chronic value NOEL for inorganic mercury identified in the literature.

Since no toxicological studies in mammalian wildlife with inorganic mercury were found in a literature search, a study with methylmercury is used for deriving a toxicity value. In a feeding study with mink (_Mustela_ _vison_), Wobeser et al. (1976 as reported in Kucera 1983) found that a level of 1.1 ppm (as methylmercury) resulted in accumulation of mercury in the brain (7.1 - 9.3 mg/kg) and signs of poisoning were evident. A toxicity value for mammalian wildlife of 0.11 is derived by dividing this value by a safety factor of 10 to convert from a LOEL to NOEL.

6.5.1.8 Nickel

The adverse effects of nickel in aquatic organisms include alteration of cell membranes, formation of precipitates on gills, hematological effects and reproductive impairment. Toxicity of nickel to freshwater organisms decreases with increasing water hardness. USEPA (1986) has proposed four-day and one-hour concentration criteria not to exceed the value given by $e^{(0.8460[\ln(hardness)]+1.1645)}$ and $e^{(0.8460[\ln(hardness)]+3.3612)}$ respectively, more than once every three years. For water with a hardness of 200 mg/liter, the four-day and one-hour criteria correspond to 280 and 2,500 ug/liter, respectively. For marine waters, the four-day and one-hour criteria are 8.3 and 75 ug/liter, respectively.

Few data are available on the toxic effects of nickel in birds. In a feeding study with mallard ducklings fed 0, 200, 800, or 1,200 ppm dietary nickel from day 1 to day 90 of age, neurological effects were observed in the highest dose

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group within 14 days of dosing (Cain and Pafford 1981). The weights of the ducks in the highest dose group were significantly decreased at 28 days of age, and the weight/length ratio of females in the 800 ppm group were significantly different from controls at days 30 and 60. A NOEL of 200 ppm can be identified for this study. No information was available on nickel toxicity in raptor species or in other species at a concentration lower than the duck NOEL.

A chronic toxicity value for mammalian wildlife of 5 mg/kg/day was derived based on information from the IRIS database (USEPA 1988b). The basis of this value was a 2 year feeding study in rats which reported a NOEL of 5 mg/kg/day.

6.5.1.9 Silver

Silver is one of the most toxic metals to aquatic organisms, and was particularly toxic to development stages of rainbow trout (Birge et al. 1981). Water hardness has an antagonistic effect on acute toxicity of silver. USEPA (1986) has established a criterion for silver not to exceed the value given by $e^{(1.72[\ln(hardness)]-6.52)}$ at any time. At a water hardness of 200 mg/liter, the acute criterion is 13 ug/liter. Available data indicate that chronic toxicity to freshwater aquatic life may occur at concentrations as low as 0.12 ug/liter. In marine waters, USEPA (1986) recommended that concentrations should not exceed 2.3 ug/liter at any time. Although no measured bioconcentration factors were available for silver, Chapman et al. (1968) estimated a BCF of 3,080 for edible portions of freshwater fish.

6.5.1.10 Zinc

Zinc is an essential trace element for aquatic organisms, and is important to cell growth and differentiation. Exposure to sublethal concentrations of zinc causes extensive edema and necrosis of liver tissue. Death results from gill necrosis and hypoxia. Zinc toxicity is dependent on water hardness. USEPA (1986) has proposed that the freshwater one-hour concentration should not exceed the value given by $e^{(0.8473[\ln(hardness)]+0.8604)}$ and the 4-day average should not exceed the value given by $e^{(0.8473[\ln(hardness)]+0.7614)}$. At a water hardness of 200 mg/liter, the one-hour and four-day criteria are 210 and 190 ug/liter, respectively. In marine waters, USEPA (1986) recommended one-hour and four-day criteria of 95 and 86 ug/liter, respectively.

The most sensitive aquatic vertebrate species tested in acute assays is the rainbow trout with a 96-hour LC_{50} of 90 ug/liter (Garton 1972); however, a 7-day EC_{50} of 30 ug/liter based on growth inhibition was reported for the green alga Selanastrum capricornutum (Bartlett et al. 1974). In chronic studies a maximum acceptable toxicant concentration of 47 ug/liter was reported for both Daphnia magna and the flagfish Jordanella floridae (Biesinger and Christensen 1972, Spehar 1978). A whole body bioconcentration factor of 432 was reported for the flagfish following 100 days of exposure (Spehar 1978).

NAS (1980) reviewed the toxic effects of zinc in livestock and poultry. Zinc poisoning has been reported in cattle. In one outbreak, poisoning was caused

by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited severe enteritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. In pigs given dietary zinc at concentrations greater than 1000 ppm, decreased food intake and weight gain were observed. At dietary levels greater than 2000 ppm, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints.

Mallard ducks fed at a level of 3,000 - 12,000 ppm became anemic, lost weight, and most died. A maximum tolerable dietary level of 1,000 ppm was recommended by NAS (1980).

A chronic oral mammalian toxicity value of 9.5 mg/kg/day was derived. The basis of this value was a 14 month study in which mice received drinking water containing 500 ppm zinc as zinc sulfate (Aughey et al. 1977 as reported in NAS 1980). While no toxic signs occurred at an estimated dose of 95 mg/kg/day (converted from 500 ppm in drinking water with values of 0.03 kg body weight and 0.0057 liter daily water intake (USEPA 1985f)), there were histological changes in the adrenal cortex and pancreas. A safety factor of 10 is used to convert from this LOEL to a NOEL.

6.5.1.11 <u>Cyanide</u>

Cyanide has been shown to cause toxic effects in both aquatic and terrestrial animal species by similar mechanisms. Following acute exposures to high doses, cyanides exert their toxic effects by inhibiting cellular respiration. Following chronic exposures to lower doses, disruption of metabolic processes other than cellular respiration may be responsible for cyanide's toxic effects.

At high concentrations, cyanide has induced death in aquatic invertebrates and fish following acute exposures, and following chronic exposures, can decrease reproduction, impair swimming ability, increase respiration, disrupt osmo- and iono-regulation, and induce histopathological effects in fish. USEPA (1986) established a freshwater four-day average criterion of 5.2 ug/liter and a one-hour average criterion of 22 ug/liter for cyanide. For marine waters, USEPA (1986) reported a criterion of 1 ug/liter both as a four-day and one-hour average concentration.

Cyanide is acutely toxic to mammals. Median lethal dosages between 3 and 4 mg/kg body weight have been reported for mice and rats (USEPA 1985g). Cyanide has caused death and toxicosis in cows and goats acutely exposed to large amounts of cyanide in forage, but dose-response data in these species are limited. In wild birds, cyanide has caused death at dosages between 4 and 21 mg/kg (Wiemeyer et al 1986). Carnivorous birds were more sensitive to the acute effects of cyanide than birds that were primarily herbivorous. Data are

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limited on the toxic effects of cyanide following chronic exposures. Cyanide has induced physiological changes in rats following long-term dietary exposure to concentrations of 73 ppm, and has caused toxic effects in piglets born to pigs exposed during gestation to cyanide in the diet at concentrations of approximately 227 ppm.

No studies have been reported showing the bioconcentration or biomagnification of cyanide in aquatic or terrestrial ecosystems.

6.5.2 Organic Chemicals

6.5.2.1 Polycyclic Aromatic Hydrocarbons

Available data on the aquatic and terrestrial toxicity of polycyclic aromatic hydrocarbons (PAHs) are limited. PAHs have been observed to cause death, teratogenesis, mutagenesis, and tumor formation in aquatic organisms. In general, PAH concentrations that are acutely toxic to aquatic species are several orders of magnitude higher than the concentrations found in even the most polluted waters (Neff 1979). A 96-hour LC50 of 820 ug/liter was reported for fluorene exposure in rainbow trout (Finger et al 1985). Following longer-term exposures, the primary adverse effect of some PAHs is tumor development. Schultz and Schultz (1982) reported liver neoplasms in approximately 44% of two species of minnows exposed 6 hours/week for 5 weeks to 5,000 ug/liter of 7,12-dimethylbenzanthracene.

In terrestrial species, the primary toxic effect of exposure to some PAHs is tumor development. Chronic oral doses of 7,12-dimethylbenzanthracene of 0.04 ug/kg have induced cancer in laboratory rodents (Lo and Sandi 1978). Much higher doses have induced cancer following short-term exposures. Food consumption in deer mice and house mice was decreased following oral exposure for 5 days to 825 or 1,213 mg/kg of 2-methoxynaphthalene. In birds, exposure to diets containing various PAHs at a total concentration of 4,000 ppm caused increased liver weights and increased blood flow to the liver (Patton and Deiter 1980).

PAHs bioaccumulate and bioconcentrate to some degree in aquatic species. A bioconcentration factor of 920 has been reported for rainbow trout continuously exposed for 10 days to 0.4 ug/liter of benzo(a)pyrene (Gerhart and Carlson 1978). PAHs do not accumulate in mammalian adipose tissue.

6.5.2.2 Polychlorinated Biphenyls

In fish, PCBs have caused death, behavioral abnormalities, increased locomotor activity, decreased success in capturing food, neurochemical alterations, disrupted osmoregulation, and liver and thyroid effects. Reproductive effects also have been linked to PCB exposure. USEPA (1986) has established four-day and one-hour concentration criteria for PCBs in freshwater of 0.014 and 2.0 ug/liter not to be exceeded more than once every 3 years. These criteria are protective of fish but were designed specifically to protect mink (Mustela vison) that eat fish from PCB-contaminated waters. In marine waters, USEPA

(1986) reported four-day average criterion of 0.03 ug/liter and a one-hour
average criterion of 10 ug/liter. In 96-hour acute assays using largemouth
bass and redear sunfish (Lepomis microlophus) exposed to Capacitor 21 (a PCB
mixture), LC_{50} values of 2.3 and 19 ug/liter, respectively, were reported
(USEPA 1980b). Sediments can act as a source of PCBs; tissue/sediment ratios
generally exceeded one (Field and Dexter 1988). Field and Dexter (1988) cite
studies that show reproductive impairment in fish when gonad concentrations
exceeded 1 ppm.

PCBs can cause death and a variety of sublethal effects in birds exposed to
the chemical via the diet. Sublethal effects following chronic exposures
include enzyme induction, porphyria, altered vitamin A metabolism, alteration
of the thyroid and cardiac, behavioral, and hormonal effects. There is
significant variability in the species sensitivity to the reproductive effects
of PCBs. No reproductive effects were observed in mallards exposed for 12
weeks during egg laying to 150 ppm Aroclor 1252 (Haseltine and Prouty 1980),
or in mallards exposed to 25 ppm Aroclor 1254 for at least a month before egg
laying (Custer and Heinz 1980). However, mourning doves (Zenaida macroura)
exposed to 10 ppm Aroclor 1254 for 28 days in the food experienced delayed
reproduction and a decrease in the number of eggs laid (Koval et al. 1987).
American kestrel exposure to 10 ppm Aroclor 1254 in food potentiated increased
egg shell thinning and subsequent breakage of eggs caused by DDE (Lincer
1972). No reproductive effects were observed in screech owls (Otus asio)
exposed for 8 weeks before the onset of egg laying to 3 ppm Aroclor 1248 in
the diet (McLane and Hughes 1980). Chickens appear to be the most sensitive

species to the reproductive effect of PCBs, with effects noted at dietary levels of 8 to 10 ppm. A NOEL of 150 ppm is identified for ducks based on the Haseltine and Prouty (1980) study in mallards. A NOEL of 3 ppm is identified for raptor species based on the McLane and Hughes (1980) study in screech owls.

These levels may not be sufficiently protective of other terrestrial species, however. Mink (Mustela vison) are particularly susceptible to the reproductive effects of PCBs. Mink fed approximately 0.2 mg/day PCBs in the form of residues in Great Lake carp failed to reproduce (Hornshaw et al. 1983), and mink fed contaminated beef from cows fed Aroclor 1254 developed reproductive complications at residue levels as low as 0.64 ppm (Platanow and Karstad 1973). Dietary concentrations only slightly higher than the latter value can be lethal to mink. A dietary LC_{50} of 6.7 ppm has been reported for Aroclor 1254 (Ringer 1983).

6.5.2.3 Dieldrin

Dieldrin is toxic to aquatic species at relatively low concentrations. Invertebrate acute toxicity occurs at concentrations ranging from 5 to 740 ug/liter. Rainbow trout (Salmo gairdneri) are the most sensitive vertebrate aquatic species tested. The USEPA (1986) criteria stated that, in fresh waters, the four-day average concentration should not exceed 0.0019 ug/liter while the one-hour average concentration should not exceed 2.5 ug/liter more than once every three years. For marine waters, USEPA (1986) established a

four-day and one-hour average criteria of 0.0019 ug/liter and 0.71 ug/liter, respectively.

Dieldrin toxicity to avian species has been summarized by Hill et al. (1975) and Hudson et al. (1984). In bobwhite and Japanese quail hatchlings 14 days old, 8- day $LC_{50}s$ of 37 and 62 mg/kg are reported. For 10-day old ring-neck pheasants an 8-day LC_{50} of 58 mg/kg was reported. In 5-day and 10-day old mallard hatchlings, 8-day $LC_{50}s$ of 153 and 169 mg/kg respectively were reported. Tests using six month old mallards reported an LD_{50} of 381 mg/kg. The most sensitive species reported was the California quail, with an LD_{50} of 9 mg/kg at 7 months.

No data were available concerning the toxicity of dieldrin to terrestrial plant species.

6.5.2.4 Volatile Organic Chemicals

Information on the aquatic toxicity of the volatile organic chemicals of concern at the SCP site is limited almost exclusively to data on acute lethality of the compounds. In general, the volatile organic chemicals are acutely toxic only at very high concentrations. For example, the acute toxicity LOEL for the chloroethylenes range from 11,600 ug/liter for 1,1-dichloroethylene to 5,280 ug/liter for tetrachloroethylene (USEPA 1986). Chloroform is slightly less toxic with an acute toxicity LOEL of 28,900 ug/liter. A 96-hour LC_{50} of 52,800 ug/liter has been reported for 1,1,1-

trichloroethane (Alexander et al. 1978). Information on the acute toxicity of 1,1-dichloroethane and chlorobenzene is lacking but, based on structure activity relationships, acute toxicity is likely to be similar to that of the chemicals discussed above.

Effects following chronic exposures will occur at concentrations lower than those producing acute effects, but quantitative dose-response information is limited. A chronic toxicity LOEL of 1,240 ug/liter has been reported for chloroform (USEPA 1986), and chlorobenzene induced mortality and teratogenesis in rainbow trout exposed to only 13 ug/liter during embryo-larval tests (Black et al. 1982).

Chronic oral mammalian toxicity values were derived for several of these compounds. For methylene chloride, a value of 5.85 mg/kg/day was derived based on information in the IRIS database (USEPA 1988b). A NOEL of 5.85 mg/kg/day from a 2 year study with rats was the basis for the toxicity value. For chloroform, a chronic oral mammalian toxicity value of 1.29 mg/kg/day was derived based on information from the IRIS database (USEPA 1988b). A LOEL of 12.9 mg/kg/day from a chronic study with dogs was the basis. A safety factor of 10 for conversion from a LOEL to a NOEL was applied. A chronic oral mammalian toxicity value of 1.7 mg/kg/day was derived based on information from the IRIS database (USEPA 1988b). This was based on a NOEL of 17 mg/kg/day from a subchronic study with rats. A safety factor of 10 was applied for conversion from a subchronic to chronic value. Insufficient

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information was available to derive toxicity values for the other volatile organic chemicals.

6.6   ECOLOGICAL RISK CHARACTERIZATION

The ecological risk evaluation is similar to human risk evaluation, in that exposure assumptions and toxicological data are combined with site data to estimate risk.   However, nonhuman receptors vary greatly in physiology and behavior, and thus it is difficult to quantify risk.   In the absence of quantitative values, validated exposure models, and other data, this ecological risk assessment will be largely restricted to a qualitative discussion of potential risks present, and how these risks might affect individuals of a species, total populations, and the ecosystem as a whole.

6.6.1   Potential Risks to Aquatic Life

Surface Water.   By direct comparison between the ambient water quality criteria (AWQC) or lowest-observed-effect levels and surface water concentrations, the potential hazards to fish and other aquatic life can be evaluated.   To date, only a limited sampling program has been conducted at the SCP site.   Four surface water samples were taken from Peach Island Creek: one upstream, one adjacent to the site, one slightly downstream, and one further downstream at the confluence of the creek with Berry's Creek.   In Table 6-1, concentrations in the upstream sample, in the higher of the two samples near the site, and in the downstream sample are presented separately.

In Table 6-1, surface water chemical concentrations were compared with USEPA (1986) freshwater and marine ambient water quality criteria. Peach Island Creek has a salinity of 4.2 parts per thousand (ppt or g/kg) adjacent to the site; freshwater has a salinity of less than 0.5 ppt (Odum 1971) while saltwater usually has salinity levels of 20 ppt or greater. In estuarine waters, USEPA recommends that concentrations be compared against the lower of the freshwater and marine AWQC (T. Purcell, USEPA Office of Water Regulations and Standards, personal communication, January 13, 1989). As shown in Table 6-1, surface water concentrations of the organic chemicals are all lower than the lowest observed effect levels (LOELS) for which toxicity data are available, with most being several orders of magnitude lower. It can be concluded, therefore, that based on the available toxicity information, the water concentrations of these organic chemicals of concern in Peach Island Creek (based on the limited sampling program undertaken) are not likely to produce adverse effects in aquatic populations.

Concentrations of copper upstream (100 ug/liter), near the site (29 ug/liter), and at the confluence with Berry's Creek (27 ug/liter) all exceed the marine acute and chronic AWQC of 2.9 ug/liter. Concentrations of mercury at all sampling stations exceed the USEPA (1986) chronic AWQC for freshwater (0.012 ug/liter) or marine waters (0.025 ug/liter). Acute AWQC for mercury (2.1 ug/liter - marine; 2.4 ug/liter - fresh) are exceeded at the upstream station (4.8 ug/liter). The chronic AWQC for nickel in marine waters (8.3 ug/liter) is exceeded at all three locations (27-57 ug/liter). Marine acute (95

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ug/liter) and chronic (86 ug/liter) criteria for zinc are exceeded at all three locations (150 - 370 ug/liter). While chromium was not detected at the two stations near the site, the maximum concentrations upstream (56 ug/liter) and downstream (28 ug/liter) both exceed the chronic (11 ug/liter) and acute (16 ug/liter) freshwater AWQC and the chronic marine AWQC (50 ug/liter) for chromium (VI). Since only total chromium was measured, the percentage that is chromium (VI) cannot be ascertained.

These data indicate that it is likely that the presence of these concentrations of inorganic chemicals will present risks of adverse effects to aquatic life in Peach Island Creek. Due to the tidal nature of Peach Island Creek, concentrations at the upstream station cannot be used as a measure of background (or non-site related) concentrations, since chemicals released from the site may be transported upstream by tidal movements.

Sediment. Sediment concentrations are compared with estimated USEPA (1988a) interim sediment criteria values to determine the potential hazards present to aquatic life exposed to sediments (Table 6-2). Sediment samples were obtained at the same locations as the surface water, and they will be evaluated in the same way by comparison with estimated interim sediment criteria.

USEPA's Office of Water has proposed use of the Equilibrium Partitioning Approach (EP) as an interim method for calculation of site specific sediment criteria (USEPA 1988a). The EP approach involves the use of partition

TABLE 6-2

COMPARISON OF MAXIMUM SEDIMENT CONCENTRATIONS
WITH SEDIMENT QUALITY CRITERIA

	Shallow Sediment Concentrations (mg/kg)			Sediment Quality Criteria (mg/kg) (a)
	Upstream	Near Site (Max. of site and 100 ft. downstream values)	Confluence w/ Berry's Creek	Mean
Semi-Volatile Chemicals				
1,2,4-Trichlorobenzene	0.525	ND	ND	NA
Bis(2-ethylhexyl)phthalate	108	32.6	2.92	8,180
Di-n-butyl phthalate	2.35	ND	ND	15,980
Di-n-octyl phthalate	ND	0.6	ND	3.38E+08
PAHs (b)				
- Acenapthene	ND	0.166	ND	73.30
- Benzo(a)pyrene	ND	0.148	ND	106.0
- Chrysene	ND	0.332	ND	39.3
- Fluoranthene	0.928	0.374	ND	188.0
- Fluorene	0.536	0.202	ND	1.54
- Naphthalene	1.33	1.23	ND	141
- Phenanthrene	1.82	0.712	ND	13.9
- Pyrene	0.916	0.339	ND	131.0
Pesticides/PCBs				
Dieldrin	ND	11	ND	0.013
PCBs (c)				
- Aroclor 1242	21	55	ND	1.39
- Aroclor 1248	ND	ND	19	0.64
- Aroclor 1254	ND	ND	5.2	1.93
- Aroclor 1260	10	6	ND	9.30
Total PCBs (d)	31	61	24.2	1.39

(a) Sediment Quality Criteria were determined by EPA (1988a) Equilibrium Partitioning Approach by multiplying AWQC or other toxicity value (e.g., LOEL) by the organic carbon partitioning coefficient (Koc) and the fraction organic carbon (assumed to be 10%).

(b) For PAHs, where no toxicity data are available, the value for benzo(a)pyrene (EPA 1988a) is substituted to obtain a rough estimate of a sediment quality criterion.

(c) For Aroclor 1242, 1248, and 1260, the toxicity value for Aroclor 1254 (EPA 1988a) is used to obtain a rough estimate of a sediment quality criterion.

(d) Sediment Quality Criteria for Aroclor 1242 used since it contributed the bulk of the PCBs in the upstream and near site areas.

NA = Insufficient toxicity data available to determine sediment quality criteria.
ND = Not detected.

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coefficients in one of two ways: (1) to derive interim Sediment Quality Criteria based on Ambient Water Quality Criteria (AWQC), or (2) to calculate sediment concentrations at a site based on surface water concentrations, and then compare these concentrations with AWQC. Since actual sediment concentrations were measured, these measurements were compared with interim Sediment Quality Criteria derived from AWQCs and available organic-carbon partition coefficients (Kocs). In order to determine site-specific SQC, a value of 0.10 for f_{oc} was used based on the observation that sediments were "waterlogged organic silt" (Dames and Moore 1990).

For PCBs, the use of the EP approach may not provide sediment quality criteria sufficiently protective of aquatic life. Recently, Field and Dexter (1988) reviewed the available literature on PCB sediment toxicity and suggested that a PCB sediment level of 0.1 mg/kg may be protective of aquatic life, although action levels derived for specific sites may vary from this level. The level was based on observed toxicity in aquatic life at organism PCB tissue concentrations between 0.1 mg/kg and 1 mg/kg and a sediment to organism concentration ratio of 1:1. Action levels based on an assumed sediment to organism ratio of 1:1 may underestimate the concentration in resident organisms, particularly organisms in higher trophic levels (Oliver and Niimi (1988), McLeese et al. (1980), Malins et al. (1980), Larson (1984), NMFS (1987)). Therefore, as Field and Dexter (1988) state, a PCB sediment level of 0.1 mg/kg may not be protective of all aquatic species. However, because data are not available to calculate more precise sediment to organism concentration ratios, the 0.1 mg/kg sediment level suggested by Field and Dexter (1988) will

be used as a preliminary action level for the protection of aquatic life in this risk assessment.

Concentrations of PCBs in sediments will be compared with both the sediment quality criteria and the 0.1 mg/kg preliminary action level.

As shown in Table 6-2, dieldrin concentrations in sediments near the site (11 mg/kg) exceed the sediment quality criteria (for sediment f_{oc} of 0.10) of 0.013 mg/kg by nearly three orders of magnitude. Concentrations of PCBs (Aroclor 1242, 1248, and 1254) exceed sediment quality criteria by factors of 2.7 to 40. The sediment quality criteria of 9.3 mg/kg for Aroclor 1260 was slightly exceeded at the upstream site (10 mg/kg) but not at the stations near the site (maximum of 6 mg/kg). Concentrations of Aroclors 1242, 1248, 1254, and 1260 exceed the 0.1 mg/kg action level (Field and Dexter 1988) by factors of 52 to 550.

Whereas no sediment quality criteria approach has been established for metals, the risks of metal contaminants in sediments can be assessed by comparing on-site concentrations with concentrations tested in laboratory bioassays. Few laboratory studies measure sediment metal concentrations. Such comparisons are further complicated by differences in sediment f_{oc}, pH, and redox potential which affect the bioavailability and toxicity of metals (Hamelink 1980; Besser and Rabeni 1987). In a laboratory study, Cairns et al. (1984) reported that 10-day LC_{50}s for aquatic invertebrates ranged from 857 to 2,296 mg copper/kg sediment. For <u>Daphnia magna</u>, these authors reported 2-day LC_{50}s

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of 681 and 937 mg/kg. The copper sediment concentrations were 9,510 mg/kg upstream, 2,000 mg/kg near the site, and 861 mg/kg at the confluence with Berry's Creek. The laboratory studies were performed in freshwater sediments while the site sediments are estuarine. In addition, f_{oc}s in the Cairns et al. (1984) laboratory study were 0.018 and 0.03 while the site sediments, described as "organic silt" by Dames and Moore (1990), may have a higher f_{oc}. Such differences may strongly influence bioavailability and toxicity. Nevertheless, the presence of copper at the upstream and near site locations does exceed reported LC_{50} values and may pose a hazard to aquatic invertebrates.

While no other similar laboratory studies were found for the other metals, there is evidence suggesting that sediment concentrations of cadmium, lead, mercury, and zinc may pose risks to aquatic life. The apparent effect threshold (AET) sediment concentration is the sediment concentration above which a statistically significant adverse effect in a biological test is expected (e.g., sediment toxicity test with a benthic invertebrate) (PTI 1987). AETs have been developed for Puget Sound based on sediment toxicity tests with the marine amphipod, Rhepoxynius abronius as follows: arsenic -- 93 mg/kg; cadmium -- 6.7 mg/kg; lead 700 mg/kg; mercury -- 2.1 mg/kg, and zinc -- 870 mg/kg. While conditions such as temperature, salinity, flushing rate, and organic carbon content are very different in Puget Sound than in Peach Island Creek, these data do provide a benchmark to evaluate sediment contaminant concentrations. A comparison of sediment concentrations collected near the site, upstream, and downstream with Puget South AETs indicates that

sediment concentrations exceeded the AETs for cadmium, mercury, and zinc. For San Francisco Bay, Chapman et al. (1987) reported an AET of 300 mg/kg for lead. This value is exceeded by the sediment concentrations at the near-site, upstream, and downstream stations.

It is emphasized that exceedence of these AET values, which are based on data from bodies of water very different from that at the site, provide only suggestive evidence of potential risks to aquatic life.

6.6.2 Potential Risks to Waterfowl

Risks to birds may be evaluated by comparing estimated concentrations of chemicals in food (using available bioconcentration factors or plant uptake factors) to the dietary concentrations identified as critical toxicity values. Estimated dietary concentrations that are below a NOEL for a chemical are not likely to pose a risk to birds. Dietary concentrations that are near, equal to or greater than a LOEL for a chemical may pose a risk to the animals.

For two chemicals, dieldrin and arsenic, only a dietary LD_{50} is available. In evaluating the potential effects of pesticides, USEPA analyzed a subset of available dose-response data and suggested that if the estimated dose is less than one-fifth of the LD_{50} for nonendangered species, no acute hazard can be presumed (Urban and Cook 1986). This approach is adopted for this risk assessment. The estimated dietary concentrations of the chemicals of concern are usually compared to one fifth of the LD_{50} value for the chemical; however,

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for endangered species such as the pied-billed grebe, a more conservative value of one-tenth of the LD_{50} value is used.

In this assessment, the potential risk associated with ingestion of contaminated aquatic invertebrates is calculated for the endangered pied-billed grebe. Dietary intakes are estimated by calculating the amount of food ingested each day and the concentration of chemical in the food. For the purposes of this assessment, it is assumed that chemicals accumulate only in the animal portion of the diet, although it is likely that some contaminants also accumulate in plants.

In determining the amount of food consumed, it must be noted that the amount of food consumed is dependent upon the intensity of activity of the animal. Smaller birds, because of their intense metabolism, are voracious feeders. A hummingbird may consume up to 100% of its body weight (approximately 3 g) every day (Hickman et al. 1979). Larger birds, such as the domestic chicken (approximately 1.9 kg), consume only approximately 3.4% of their body weight per day. To determine the dose for the pied-billed grebe, it is assumed that the grebe consumes daily an amount of food equal to 10% of its body weight.

Using an average weight of 0.34 kg for male and female grebes (Welty 1982), a daily intake of 0.034 kg is determined, and of this it is assumed that 80%, or 0.027 kg, is animal food based on discussion in Martin et al. (1951). For this assessment it is assumed that the grebe's food consists entirely of aquatic invertebrates from Peach Island Creek. The concentration of chemical

in the food is estimated using bioconcentration factors (BCFs) for aquatic invertebrates derived from the literature, shown in Table 6-3. Concentrations in the food are derived by multiplying the water concentration by the BCF (Table 6-4). The amount (mg) of chemical ingested from animal food is then determined by multiplying the concentration in the food by 0.027 kg/day. This chemical intake is then divided by the total amount of food ingested daily (0.034 kg) to determine the concentration of chemical in the entire diet (plant and animal). These intake values are compared with critical dietary toxicity values in Table 6-5.

The dietary chemical concentrations estimated for the pied-billed grebe shown in Table 6-5 exceed the toxicity values derived for dieldrin, cadmium, copper, lead, mercury, nickel, and zinc. There are considerable uncertainties associated with these estimated dietary concentrations since they are based on: (1) limited sediment sampling data, (2) estimation of water concentrations by multiplying sediment concentrations by a distribution coefficient, and (3) use of short-term invertebrate bioconcentration factors. Uncertainties also exist in the toxicity values which were derived from studies with other species. In addition, it has been assumed that 100% of the diet is from Peach Island Creek. While definitive conclusions are not possible, the potential for adverse effects to the pied-billed grebe, and other species, cannot be precluded.

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TABLE 6-3

INVERTEBRATE BIOCONCENTRATION FACTORS
FOR CHEMICALS OF CONCERN

	Invertebrate Bioconcentration Factor (BCF)	Basis	Reference
PAHs (a)	82,200	3-day bioconcentration in snails	Eisler 1987b
Dieldrin	1,800	bioconcentration in aquatic molluscs	Verschueren 1983
PCBs	6,200	whole body bioconcentration in amphipods (Gammarus sp) exposed for 21 days to Aroclor 1254	EPA 1980d
Arsenic	17	whole body bioconcentration by snails (Helisoma campanulatum) for 28 days of exposure	EPA 1985a
Cadmium	4,190	28-week whole body bioconcentration by caddis fly (Hydropsyche botteni) nymphs	EPA 1985b
Copper	203	14 day bioconcentrations in stonefly (Pteronarcys california)	EPA 1985d
Lead	2,370	whole body bioconcentration by snails (Helisoma campanulatum) for 28 days of exposure	EPA 1985h
Mercury	2,500	7-day bioconcentration in amphipods (Gammarus sp)	EPA 1985e
Nickel	192	estimated whole body bioconcentration in cladocerans (Daphnia sp) exposed for 3.75 days	EPA 1986
Zinc	571	14 day bioconcentrations in stonefly (Pteronarcys california)	EPA 1985i

(a) Based on data for benzo(a)pyrene.

TABLE 6-4

ESTIMATED CHEMICAL CONCENTRATIONS IN AQUATIC INVERTEBRATES AND THE WHOLE DIET OF THE PIED-BILLED GREBE

	Shallow Sediment Concentrations (mg/kg)			Sediment to Water Conversion Factor (a)	Estimated Water Concentrations (mg/l)			Invertebrate BCF	Estimated Invertebrate Concentrations (mg/kg)			Whole Diet Concentration (mg/kg)		
	Upstream	Near Site (Max. of two values) (b)	Confluence w/ Berry's Creek		Upstream	Near Site (Max. of two values) (b)	Confluence w/ Berry's Creek		Upstream	Near Site (Max. of two values) (b)	Confluence w/ Berry's Creek	Upstream	Near Site (Max. of two values) (b)	Confluence w/ Berry's Creek
PAHs														
- Acenapthene	ND	0.166	ND	112	NE	1.48E-03	NE	82,200	NE	1.22E+02	NE	NE	9.66E+01	NE
- Benzo(a)pyrene	ND	0.148	ND	4,470	NE	3.31E-05	NE	82,200	NE	2.72E+00	NE	NE	2.16E+00	NE
- Chrysene	ND	0.332	ND	1,780	NE	1.87E-04	NE	82,200	NE	1.54E+01	NE	NE	1.22E+01	NE
- Fluoranthene	0.928	0.374	ND	977	9.50E-04	3.83E-04	NE	82,200	7.81E+01	3.15E+01	NE	6.20E+01	2.50E+01	NE
- Fluorene	0.536	0.202	ND	81	6.59E-03	2.49E-03	NE	82,200	5.42E+02	2.04E+02	NE	4.31E+02	1.62E+02	NE
- Naphthalene	1.33	1.23	ND	14	9.42E-02	8.71E-02	NE	82,200	7.74E+03	7.16E+03	NE	6.15E+03	5.69E+03	NE
- Phenanthrene	1.82	0.712	ND	148	1.23E-02	4.81E-03	NE	82,200	1.01E+03	3.96E+02	NE	8.04E+02	3.14E+02	NE
- Pyrene	0.916	0.339	ND	955	9.59E-04	3.55E-04	NE	82,200	7.89E+01	2.92E+01	NE	6.26E+01	2.32E+01	NE
Pesticides/PCBs														
Dieldrin	ND	11	ND	1,050	NE	1.05E-02	NE	1,800	NE	1.90E+01	NE	NE	1.51E+01	NE
PCBs:														
- Aroclor 1242	21	55	ND	71	2.97E-01	7.77E-01	NE	6,200	1.84E+03	4.82E+03	NE	1.46E+03	3.83E+03	NE
- Aroclor 1248	ND	ND	19	2,400	NE	NE	7.92E-03	6,200	NE	NE	4.91E+01	NE	NE	3.90E+01
- Aroclor 1254	ND	ND	5.2	4,370	NE	NE	1.19E-03	6,200	NE	NE	7.39E+00	NE	NE	5.87E+00
- Aroclor 1260	10	6	ND	47,900	2.09E-04	1.25E-04	NE	6,200	1.30E+00	7.77E-01	NE	1.03E+00	6.17E-01	NE
Inorganic Chemicals														
Arsenic	37	ND	34	5	7.40E+00	NE	6.80E+00	17	1.26E+02	NE	1.16E+02	9.99E+01	NE	9.18E+01
Cadmium	83.7	43.4	32	7	1.29E+01	6.68E+00	4.92E+00	4,190	5.40E+04	2.80E+04	2.06E+04	4.28E+04	2.22E+04	1.64E+04
Lead	320	520	360	900	3.56E-01	5.78E-01	4.00E-01	2,370	8.41E+02	1.37E+03	9.46E+02	6.68E+02	1.09E+03	7.52E+02
Mercury	40.6	25.4	139	10	4.06E+00	2.54E+00	1.39E+01	2,500	1.02E+04	6.35E+03	3.48E+04	8.06E+03	5.04E+03	2.76E+04
Nickel	467	110	100	40	1.17E+01	2.75E+00	2.50E+00	192	2.24E+03	5.28E+02	4.80E+02	1.78E+03	4.19E+02	3.81E+02
Zinc	3110	2320	2880	40	7.78E+01	5.80E+01	7.20E+01	571	4.44E+04	3.31E+04	4.11E+04	3.53E+04	2.63E+04	3.26E+04

(a) Using the equations: $Cw = Cs/(Koc*foc)$ for organics and $Cw = Cs/Kd$ for inorganics, based on the USEPA (1988a) Equilibrium Partitioning Approach.
(b) Maximum of measurements adjacent to site and 100 feet downstream.

ND = Not detected.
NE = Not estimated; chemical not detected at this location.

TABLE 6-5

COMPARISON OF ESTIMATED WHOLE DIET CONCENTRATIONS FOR THE
PIED-BILLED GREBE WITH SELECTED TOXICITY VALUES

	Whole Diet Concentration (mg/diet)			Toxicity Value (mg/diet)
	Upstream	Near Site (Max. of two values) (b)	Confluence w/ Berry's Creek	
PAHs				
- Acenapthene	NE	8	NE	4,000 LOEL
- Benzo(a)pyrene	NE	0.11	NE	4,000
- Chrysene	NE	1	NE	4,000
- Fluoranthene	4	2	NE	4,000
- Fluorene	27	10	NE	4,000
- Naphthalene	579	535	NE	4,000
- Phenanthrene	53	21	NE	4,000
- Pyrene	6	2	NE	4,000
Pesticides/PCBs				
Dieldrin	NE	15	NE	0.9 (1/10th LD50 of 9)
PCBs:				
- Aroclor 1242	1	3	NE	150 NOEL
- Aroclor 1248	NE	NE	2	150
- Aroclor 1254	NE	NE	0.19	150
- Aroclor 1260	0.07	0.04	NE	150
Inorganic Chemicals				
Arsenic	100	NE	92	500 (1/10th LD50 of 5,000)
Cadmium	42,800	22,200	16,400	4 LOEL
Copper	43,800	9,210	3,970	300 MTD (a)
Lead	668	1,090	752	50 NOEL
Mercury	8,060	5,040	27,600	32 NOEL
Nickel	1,780	419	381	200 NOEL
Zinc	35,300	26,300	32,600	1,000 MTD (a)

(a) Maximum tolerable dietary level recommended by NAS (1980).
(b) Maximum of measurements adjacent to site and 100 feet downstream.

NE = Not estimated; chemical not detected at this location.

6.6.3 Potential Risks to Mammalian Wildlife

Muskrat using Peach Island Creek or the wetlands as a source of drinking water may be exposed to contaminants. Muskrats weigh about 1.1 kg (Hoffmeister and Mohr 1972). While no rate of drinking water ingestion could be found for muskrats, rabbits with an average weight of 1 kg consume 0.25 liter of water per day (USDA 1988). Thus, for muskrats a value of 0.28 liter will be used, assuming the same relationship between weight and intake. This may be an overestimation, since the estuarine (4.2 parts per thousand salinity) nature of the area may cause the muskrat to obtain more of its water needs from its diet. Possible uptake of contaminants by contact with sediments and subsequent grooming cannot be quantified. Although muskrats, which are largely herbivorous (Martin et al. 1951), could obtain contaminants through the diet, concentrations in marsh plants have not been determined. Thus, only exposure from drinking water will be assessed.

The estimated doses to muskrat from drinking water ingestion are compared with toxicity values in Table 6-6. Toxicity values were not available for the other chemicals of concern in surface water: chlorobenzene, methyl ethyl ketone, toluene, 1,1,1-trichloroethane, trichloroethylene, or xylenes. As shown in Table 6-6, all of the estimated doses were at least one and often 2-3 orders of magnitude lower than the toxicity values. Therefore, it is unlikely that exposure to contaminants in drinking water will present a risk to muskrat or other mammalian wildlife.

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TABLE 6-6

COMPARISON OF ESTIMATED DOSES FOR MUSKRAT
WITH SELECTED TOXICITY VALUES

	Surface Water Concentration (ug/l)			Estimated Dose (mg/kg/day)			
	Upstream	Near Site (a)	Confluence w/ Berry's Creek	Upstream	Near Site (a)	Confluence w/ Barry's Creek	Toxicity Value (mg/kg/day)
Chloroform	ND	3.58	ND	ND	9.1E-04	ND	1.29
1,2-trans-Dichloroethylene	ND	35.2	3.91	ND	8.8E-03	1.0E-05	1.7
Methylene chloride	4.63	12.9	14.9	1.2E-03	3.2E-03	3.7E-03	5.85
Chromium	56	ND	28	1.4E-02	ND	7.0E-03	0.24
Copper	100	29	27	2.5E-03	7.2E-03	6.8E-03	6
Mercury	4.8	0.96	1.1	1.2E-03	2.4E-04	2.8E-04	0.11
Nickel	57	33	27	1.4E-02	8.2E-03	6.8E-03	5
Zinc	370	160	150	9.2E-02	4E-02	3.8E-02	9.5

(a) Maximum of measurements adjacent to site and 100 feet downstream.

ND = Not detected.

6.7 SUMMARY OF ENVIRONMENTAL RISK

Potential risks to environmental receptors have been evaluated based on the results of site monitoring data, a review of the toxicity of chemicals of concern, and estimates of exposure. Risks have been characterized by comparing concentrations with federal criteria and by comparing estimated exposure with toxicity values based on sceintific literature. The assumptions used in estimating exposures and deriving toxicity values have been described in this section. The hazards from exposures of aquatic and terrestrial life to chemicals in surface water and sediments of Peach Island Creek have been assessed. Since the site is sparsely vegetated, has been disturbed by human activity, and no terrestrial mammals were observed, this assessment has not considered on-site soils to be a potential source of contaminant exposure for terrestrial animals. In addition, the man-made fill material which makes up a large portion of the site's surface is not favorable for plant growth, as evidenced by the sparse plant growth, consisting largely of common grasses and weeds. In order for other natural vegetation to become established, soil conditions on site would have to be at least partially restored. Thus, this assessment did not consider potential phytotoxic effects from chemicals in on-site soils. The results of the environmental risk assessment are summarized below.

The hazards to aquatic life were evaluated by comparing surface water and sediment concentrations with USEPA ambient water quality criteria (AWQC), sediment quality criteria (SQC), and other toxicity values. Risks to aquatic

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life may result from exposure to chemicals in surface water since ambient water quality criteria were exceeded for copper, mercury, nickel, and zinc. Risks to aquatic life from chemicals in sediments are also likely to occur since (1) dieldrin and PCB concentrations exceed sediment quality criteria by up to three orders of magnitude, (2) copper concentrations in sediments exceed concentrations that were shown to be lethal in short-term laboratory tests with invertebrates, and (3) concentrations of cadmium, lead, mercury and zinc exceed levels that have produced biological effects in sediment toxicity tests conducted in west coast estuaries. In addition, PCB concentrations in sediments exceed the 0.1 mg/kg preliminary action level proposed by Field and Dexter (1988) by factors of 52 to 550. Based on these comparisons it is apparent that the presence of site-related contaminants poses a substantial risk to populations of aquatic life that may inhabit Peach Island Creek.

Additionally, potential risks to the pied-billed grebe, which has an endangered breeding population in the Meadowlands not far from Berry's Creek, were evaluated. Estimated dietary concentrations of dieldrin, cadmium, copper, lead, mercury, nickel, and zinc exceed toxicity values derived from studies on birds. There are considerable uncertainties associated with these estimated dietary concentrations since they are based on: (1) limited sediment sampling data, (2) estimation of water concentrations by multiplying sediment concentrations by a distribution coefficient, and (3) use of short-term invertebrate bioconcentration factors. These data indicate, however, that adverse effects to this endangered breeding population might occur should they exist near the site or feed extensively from Peach Island Creek. Birds

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that frequent other, less contaminated areas for a portion of their diet, would be at less risk.

It is unlikely that mammalian wildlife such as the muskrat will suffer adverse effects from the ingestion of contaminants in surface water. Estimated doses were at least one order of magnitude below toxicity values for all chemicals of concern. Other possible routes of exposure for mammalian wildlife, such as through the diet or contact with sediments and subsequent grooming, have not been quantified because data are insufficient to estimate doses or dietary levels.

7.0 UNCERTAINTIES IN RISK ASSESSMENT

The procedures and inputs used to assess risks in this evaluation, as in all such assessments, are subject to a wide variety of uncertainties. In general, the main sources of uncertainty include:

- Environmental chemistry sampling and analysis

- Environmental parameter measurement

- Fate and transport modeling

- Exposure parameter estimation

- Toxicological data

Environmental chemistry analysis error can stem from several sources including the errors inherent in the analytical methods, chain of custody problems, or the characteristics of the matrix being sampled. For this assessment, the analytical methods chosen were all methods approved by USEPA. However, data validation was not performed. Thus, procedural or systematic errors cannot be ruled out. A split sample comparison of 27 samples, however, indicated no trends in the results of 762 analytes which were independently measured by two laboratories. In this splits analysis, there were 409 cases (54% of the total) of higher results in one laboratory and 353 cases (46%) of higher results in the second laboratory.

Another analytical problem potentially affecting the risk assessment concerns the sample detection limits attained for the chemicals of concern at the SCP site. Although certain chemicals were not detected in soils and sediments at the site, in some instances the sample detection limits may have been several times higher than reported concentrations and/or CLP detection limits. It is uncertain, therefore, whether these chemicals are present above or below a level of concern in these media. Exclusion of chemicals present at levels below the detection limit, but above the levels of concern, from the risk assessment, would underestimate the risks associated with certain exposures. On the other hand, exclusion of chemical concentrations below both the detection limit and the levels of concern would not significantly impact the risk estimates.

The sample size also affects the level of uncertainty in a risk assessment. Generally, the larger the sample size the smaller the level of uncertainty. For example, in this assessment, many samples were collected from soils at the site. In contrast, only one or two sample results were available from the bedrock aquifer.

The use of currently measured concentrations to represent potential future concentrations also contributes uncertainty, although in the conservative direction. For example, to evaluate exposures to a possible future on-site worker, current soil and ground water concentrations were assumed to persist into the future, and then throughout the worker's assumed period of exposure. For both the current and future scenarios, these exposure point concentrations

do not reflect the likely reductions in concentration over time due to migration, biodegradation, and volatilization. This approach is most important for the soil and ground water exposure pathways since the concentrations of volatile and many semi-volatile organics currently present will be reduced over time. The uncertainty associated with using current sampling data to estimate exposures both over a several year period (e.g., nearby workers) and in the future (e.g., a possible on-site worker) may be as much as an order of magnitude.

Environmental parameter measurements primarily contribute to uncertainty because little verified information is available. Lack of site-specific measurements requires that estimates be made on the basis of literature values, extrapolations from regression equations, and/or best professional judgement. In this assessment several parameter values were assumed for the air emissions and dispersion modeling because site-specific data were unavailable (e.g., site measurements of organic carbon content and bulk density of the soil).

Modelling error can arise from the use of an inappropriate model or the use of an appropriate model with inappropriate boundary conditions. In this assessment both air emissions and dispersion models were applied for the inhalation pathways. While these models were considered to be appropriate for the exposure pathways under evaluation, there are nevertheless uncertainties associated with them. For example, the Hwang volatilization model does not explicitly take into account the effects of temperature, the presence of a

stagnant boundary layer, and soil moisture on the rate of volatilization. Therefore it overestimates volatilization of soluble, low molecular weight chemicals (such as chlorinated and non-chlorinated solvents) which are diffusion limited. This leads to an overestimation of potential risks via inhalation for these chemicals. On the other hand, the model underestimates the length of exposure over which the highest flux rates occur which would underestimate potential risks. To compensate for this, air concentrations were derived only for the time period during which emissions would occur (e.g., less than five years) even though the durations of exposure were usually longer than this (e.g., 30 years for a nearby resident). A fugitive dust emission model was used to estimate airborne concentrations of suspended soils. It should be noted that some of the surface materials at the site are likely to be too large to be entrained by wind, e.g., gravel. Therefore, a limited erosion potential emissions equation was used for this risk assessment. Additionally, the model assumed that the reservoir of contaminated soil will not be depleted over time. Other uncertainties can stem from a lack of validation or verification of the models. Roughly an order of magnitude uncertainty may be associated with the use of these models to predict air concentrations in this assessment.

With respect to the exposure scenarios evaluated in this risk assessment, there are several uncertainties in determining the exposure parameters that will go into the scenario and that will ultimately be combined with toxicological information to assess risk. For example, there are a number of uncertainties regarding estimates of how often, if at all, an individual would

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come into contact with the chemicals of concern and the period of time over which such exposures would occur. For example, worker contact with surface soil was assumed to occur a specified number of days each year for the exposure period evaluated. These assumptions may yield risks which are overestimated or underestimated, depending on actual work habits. Where specific exposure parameter values were provided by USEPA (1989a) for the RME case, they were used, at the request of USEPA Region II, but were not independently verified. In addition, other assumptions used in this assessment (e.g., ingestion of one liter of water per day by a worker, 70 kg average body weight, and a 30-year exposure period for a nearby resident), are assumed to represent upper bounds of potential exposure and have been used when site-specific data are not available or when hypothetical situations are being investigated (e.g., a future on-site worker). Risks for certain individuals within an exposed population will be higher or lower depending on their actual drinking water intakes, body weights, etc.

Toxicological data error is one of the largest sources of uncertainty in this risk assessment. As USEPA notes in its Guidelines for Carcinogenic Risk Assessment (USEPA 1986b):

> There are major uncertainties in extrapolating both from animals to humans and from high to low doses. There are important species differences in uptake, metabolism, and organ distribution of carcinogens, as well as species and strain differences in target site susceptibility. Human populations are variable with respect to genetic constitution, diet, occupational and home environment, activity patterns and other cultural factors.

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To compensate for these uncertainties, conservative assumptions are used in deriving the toxicity criteria applied in this assessment. The uncertainty in these criteria may, however, account for an order of magnitude or more uncertainty in the final risk estimates, although this is biased in the conservative direction (i.e., risks may be overestimated but are unlikely to be underestimated).

In addition, a large degree of uncertainty results from the lack of EPA-approved health criteria (slope factors and reference doses) for use in quantitatively evaluating potential risks. This is particularly true for the inhalation exposure pathways, for which USEPA-approved route-specific slope factors and reference doses are not available for numerous chemicals of concern (e.g., carcinogenic PAHs, PCBs, antimony, bis(2-ethylhexyl)phthalate, cyanide, 2,4-dimethylphenol, di-n-butylphthalate). This uncertainty may result in an underestimation of risks.

A particular problem is presented by the necessity to perform risk assessments for polycyclic aromatic hydrocarbons (PAHs). PAHs occur in the environment as complex mixtures of many components with widely varying toxic potencies. Only a few components of these mixtures have been adequately characterized, and only limited information is available on potential synergistic effects of the PAH mixture. The approach adopted by USEPA (1980, 1984) and used in this report as the basis for risk assessment is to divide the PAHs into two subclasses, "carcinogenic" PAHs and "noncarcinogenic" PAHs, and to apply a cancer potency factor derived from oral bioassays on benzo[a]pyrene (B[a]P) to

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the subclass of carcinogenic PAHs. Most evidence indicates that benzo[a]pyrene is more potent than most of the other carcinogenic PAHs and usually represents only a small portion of a mixture of carcinogenic PAHs (Schmahl et al. 1977, Pfeiffer 1977); therefore, this technique will probably overestimate risk.

Uncertainty is also associated with chemical speciation of chromium for the inhalation of suspended soil pathway. Hexavalent chromium (Cr VI) is carcinogenic when inhaled while trivalent chromium is not. In this assessment, chromium on suspended soils was assumed to be in the hexavalent form. In actuality, only a fraction of the chromium present on suspended soil will be Cr VI and thus the risks for this chemical are likely to be overestimated.

There is also a great deal of uncertainty in assessing the toxicity of a mixture of differing chemicals. In this assessment, the effects of exposure to each of the contaminants present in the environmental media have initially been considered separately. However, these substances occur together at the site, and individuals may be exposed to mixtures of the chemicals. Prediction of how these mixtures of toxicants will interact must be based on an understanding of the mechanisms of such interactions. The interactions of the individual components of chemical mixtures may occur during absorption, distribution, metabolism, excretion, or activity at the receptor site. Individual compounds may interact chemically, yielding a new toxic component or causing a change in the biological availability of an existing component,

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or may interact by causing different effects at different receptor sites. Suitable data are not currently available to rigorously characterize the effects of chemical mixtures similar to those present at the SCP site. Consequently, as recommended in USEPA's Risk Assesment Guidance for Superfund (USEPA 1989a) and in USEPA's Guidelines for Health Risk Assessment of Chemical Mixtures (USEPA 1986c), chemicals present at the SCP site were assumed to act additively, and potential health risks were evaluated by summing excess cancer risks and calculating hazard indices for chemicals exhibiting carcinogenic and noncarcinogenic effects, respectively. This approach to assessing the risk associated with mixtures of chemicals assumes that there are no synergistic or antagonistic interactions among the chemicals considered and that all chemicals have the same toxic end points and mechanisms of action. To the extent that these assumptions are incorrect, the actual risk could be under- or overestimated.

As a result of the uncertainties described above, this risk assessment should not be construed as presenting absolute estimates of risks to human or environmental populations. Rather, it is a conservative analysis intended to indicate the potential for adverse impacts to occur.

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8.0 SUMMARY AND CONCLUSIONS

The SCP site is currently the subject of a Comprehensive Environmental Response, Compensation and Liability Act (CERCLA, as amended), Remedial Investigation/Feasibility Study (RI/FS). The site is classified as an enforcement lead site for which potentially responsible parties (PRPs) are conducting the RI/FS. At the request of Region II of the U.S. Environmental Protection Agency (USEPA) this Baseline Risk Assessment (BRA) was prepared under the REM III Superfund contract. It is a stand-alone document, conducted independently of the PRPs' RI and BRA efforts, but which relies on the Remedial Investigation conducted by Dames and Moore (1990) for the PRPs as a primary source of information.

8.1 SELECTION OF CHEMICALS FOR EVALUATION

Forty-three chemicals of concern which exist in the soil and/or ground water at the site (see Table 8-1) were selected for detailed evaluation in this Baseline Risk Assessment. The sampling data used in this selection process and in the evaluation were collected and analyzed as part of the Dames and Moore (1990) RI in addition to two samples collected from the bedrock aquifer in 1989 and analyzed by USEPA during continuation of the Dames and Moore RI work. The Dames and Moore (1990) RI data were obtained from the raw laboratory data reports (ETC December 1987) provided to USEPA by Dames and Moore.

TABLE 8-1

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Soil	**Human Health Risks**	
	Excess lifetime cancer risks exceeded 1E-06 (one in one million) for trespassers who may contact on-site surface soil (average and reasonable maximum cases).	Aldrin, arsenic, carcinogenic PAHs, 1,1-dichloroethane, dieldrin, PCBs, tetrachloroethylene, trichloroethylene
	Under future site use conditions, excess lifetime cancer risks exceeded 1E-06 for possible future on-site workers who may contact surface soil (average and reasonable maximum cases).	Aldrin, arsenic, benzene, bis(2-ethylhexyl)phthalate, carcinogenic PAHs, 1,1-dichloroethane, 1,2-dichloroethane, dieldrin, PCBs, tetrachloroethylene, trichloroethylene
	Under future site use conditions, excess lifetime cancer risks exceeded 1E-06 for possible future on-site construction workers who may contact subsurface soil (reasonable maximum case).	Carcinogenic PAHs, PCBs
	Adverse noncarcinogenic effects may occur among trespassers who contact surface soil under current site use and among possible future on-site workers who contact surface soil (reasonable maximum cases only).	Aldrin, dieldrin, PCBs, tetrachloroethylene, trichloroethylene
	Ecological Risks	
	Not evaluated. The site is not currently a habitat for terrestrial wildlife or abundant plant life. The extent to which this is a result of site contamination could not be determined.	
	Migration Potential	
	Contaminants present in on-site soil have migrated downward to deeper soil depths, and into the water table and till aquifers. Further migration into the bedrock aquifer has also occurred. Chemicals may also be released from soil into air via volatilization and suspension or surface soil by wind or vehicles. Chemicals may migrate via surface soil runoff into Peach Island Creek.	VOCs, PAHs, PCBs
	Violations of ARARs/Other Guidance	
	NJDEP ECRA Objectives for soil	Total volatile organics (predominantly chlorobenzene, 1,1-dichloroethane, ethylbenzene, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene, xylenes)
		Total base neutrals/acids (predominantly PAHs, phenol, butyl benzyl phthalate, 1,2-dichlorobenzene)
		PCBs
		Inorganics (predominantly cadmium, copper, lead, mercury, zinc)
	TSCA PCB Spill Policy	PCBs

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations r------ded A^^^- and ' igrat- 'n th   ironr   nav t   ecte

TABLE 8-1 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Ground Water (Water Table and Till Aquifers)	**Human Health Risks**	
	Excess lifetime cancer risks for a possible future on-site worker, assumed to regularly consume on-site ground water, were greater than 1E-06 (one in one million) for both the water table and till aquifers for the average and reasonable maximum cases.	Arsenic, benzene, bis(2-ethylhexyl)phthalate, carcinogenic PAHs, chloroform, 1,1-dichloroethane, 1,2-dichloroethane, 1,1-dichloroethylene, isophorone, methylene chloride, PCBs, 1,1,2,2-tetrachloroethane, tetrachloroethylene, trichloroethylene, vinyl chloride
	Adverse noncarcinogenic effects may occur for a possible future on-site worker assumed to regularly consume on-site ground water for both the water table and the till aquifers (average and reasonable maximum cases).	Arsenic, chlorobenzene, chloroform, 1,2-dichloroethane, 1,2-trans-dichloroethylene, lead, methylene chloride, methyl ethyl ketone, nitrobenzene, PCBs, 1,1,2,2-tetrachloroethane, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene
	Ecological Risks	
	Not evaluated; no aquatic or terrestrial wildlife species are expected to come into contact with on-site ground water.	
	Migration Potential	
	Chemicals present in the water table and till aquifers may migrate into Peach Island Creek and to deeper soil depths. The water table aquifer flow is not well defined although it appears to flow radially across the site's boundaries and downward to the till aquifer. The till aquifer appears to flow towards the northwest. Further vertical migration into the bedrock aquifer has also occurred.	VOCs, PCBs
	Violations of ARARs/Other Guidance	
	Federal MCLs and MCLGs	Benzene, 1,2-dichloroethane, 1,1-dichloroethylene, ethylbenzene, 1,1,1-trichloroethane, chloroform, chlorobenzene, trichloroethylene, vinyl chloride, arsenic, cadmium, chromium, lead, mercury
	State MCLs	Benzene, chlorobenzene, chloroform, 1,1-dichloroethylene, 1,2-dichloroethane, 1,1,1-trichloroethane, methylene chloride, 1,2-trans-dichloroethylene, xylenes, tetrachloroethylene, trichloroethylene, vinyl chloride, arsenic, cadmium, chromium, lead, mercury, PCBs
	State Ground Water Quality Standards	Benzene, chlorobenzene, chloroform, 1,2-dicholroethane, 1,1-dichloroethylene, 1,2-trans-dichloroethylene, methylene chloride, tetrachloroethylene, 1,1,1-trichlroethane, trichloroethylene, vinyl chloride, xylenes, PCBs, phenol, arsenic, cadmium, chromium, copper, lead, mercury
	Federal Ambient Water Quality Criteria for Protection of Human Health (adjusted for drinking water only)	Benzene, 1,2-dichloroethane, 1,1-dichloroethylene, 1,1,2,2-tetrachloroethane, chlorobenzene, 1,1,1-trichloroethane, chloroform, ethylbenzene, tetrachloroethylene, trichloroethylene, toluene, vinyl chloride, PCBs, phenol, arsenic, beryllium, cadmium, chromium, copper, lead, nickel

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(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

TABLE 8-1 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Ground Water (Bedrock aquifer)	**Human Health Risks**	
	Excess lifetime cancer risks for a possible future on-site worker, assumed to regularly consume on-site bedrock ground water, were greater than 1E-06 (one in one million) for the average and reasonable maximum cases.	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, trichloroethylene, vinyl chloride
	Adverse noncarcinogenic effects may occur for a possible future on-site worker, assumed to regularly consume on-site groundwater from the bedrock aquifer (average and reasonable maximum cases).	Chloroform, trichloroethylene
	Ecological Risks	
	Not evaluated; no aquatic or terrestrial wildlife species are expected to come into contact with on-site ground water.	
	Migration Potential	
	Chemicals present in the bedrock aquifer may migrate to off-site locations from which water may be used as a public water supply.	VOCs
	Violations of ARARs/Other Guidance	
	Federal MCLs and MCLGs	Chloroform, 1,2-dichloroethane, trichloroethylene, vinyl chloride
	State MCLs	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, methylene chloride, tetrachloroethylene, trichloroethylene, vinyl chloride
	State Ground Water Quality Standards	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, methylene chloride, tetrachloroethylene, trichloroethylene, vinyl chloride
	Federal Ambient Water Quality Criteria for Protection of Human Health (adjusted for drinking water only).	Chloroform, 1,2-dichloroethane, 1,1-dichloroethylene, tetrachloroethylene, trichloroethylene, vinyl chloride

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

TABLE 8-1 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Surface Water	**Human Health Risks**	
	Not evaluated due to limited sampling data. Peach Island Creek near the site is, however, currently accessible to some extent. Since access could change in the future, this pathway should be evaluated if more data become available in the future.	
	Ecological Risks	
	Adverse effects to aquatic life may occur from short- and long-term exposure to concentrations of inorganic chemicals in Peach Island Creek.	Copper, mercury, nickel, zinc
	No adverse effects are expected to occur in mammalian wildlife (such as muskrats) through ingestion of surface water.	
	Migration Potential	
	Chemicals in Peach Island Creek may be transported both up and down stream although the magnitude of impact of the site is difficult to determine due to complex tidal nature of the creek and availability of only limited sampling results.	Ethylbenzene, xylenes, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene, PCBs, dieldrin, bis(2-ethylhexyl)phthalate, 1,2-dichlorobenzene
	Exceedances of ARARs/Other Guidance	
	State Surface Water Quality Standards	PCBs

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

TABLE 8-1 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Sediment	**Human Health Risks**	
	Not evaluated.	
	Ecological Risks	
	Adverse effects to aquatic life may occur from short- and long-term exposure to inorganic and organic chemicals in sediments.	Dieldrin, PCBs, cadmium, copper, lead, mercury, zinc.
	Adverse effects may occur in water fowl (including endangered species) by ingesting contaminated invertibrates. There are considerable uncertainties (e.g., in calculated interstitial water concentrations and bioconcentration factors) in these estimates.	Dieldrin, cadmium, copper, lead, mercury, nickel, zinc.
	Migration Potential	
	Chemicals on-site have migrated into Peach Island Creek sediment, although the magnitude of impact is difficult to determine due to complex tidal nature of the creek and availability of only limited sampling results.	Ethylbenzene, xylenes, tetrachloroethylene, toluene, 1,1-trichloroethane, trichloroethylene, PCBs, dieldrin, bis(2-ethylhexyl)phthalate, 1,2-dichlorobenzene.
	Violations of ARARs/Other Guidance	
	Proposed NOAA sediment action level for protection of aquatic life.	PCBs

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

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TABLE 8-1 (continued)

SUMMARY OF SCP SITE ENDANGERMENT ASSESSMENT (a)

Environmental Medium	Results	Predominant Chemicals (b)
Air	**Human Health Risks** ------------------- Excess lifetime cancer risks for nearby workers who may inhale volatilized organics and suspended soil transported from the site exceeded 1E-06 (one in one million) (reasonable maximum case only). Adverse noncarcinogenic effects are not expected to occur.	Chromium, 1,1-dichloroethylene, vinyl chloride
	Excess lifetime cancer risk for possible future on-site workers who may inhale volatilized organics and suspended soil exceeded 1E-06 (one in one million) for both the average and reasonable maximum cases. Adverse noncarcinogenic effects are not expected to occur.	Chloroform, chromium, 1,1-dichloroethylene, methylene chloride, trichloroethylene, vinyl chloride
	Ecological Risks ---------------- Not evaluated; no terrestrial mammals observed on-site. Ambient air exposures are not likely to result in significant exposures.	
	Migration Potential --------------------- Chemicals released into air from the site (via volatilization and suspension of surface soil) may migrate off-site.	VOCs, PAHs, PCBs
	Violations of ARARs/Other Guidance --------------------------------- Not available except for lead and its ARAR was not exceeded.	

(a) The information in this table is abbreviated from the text of the risk assessment, and thus should only be used in conjunction with this document.
(b) For each chemical listed above, the excess lifetime cancer risk exceeded 1E-06, and/or adverse noncarcinogenic effects may occur, and/or the concentrations exceeded ARARs, and/or migration in the environment may be expected.

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The selected chemicals fall within the following classes of contaminants: VOCs (17), pesticides (2), PCBs, semi-volatile compounds (12) and inorganic chemicals (11).

The highest concentrations for almost all the chemicals of concern listed in Table 8-1 are found in the soil and/or ground water located above the clay lens at the site. The levels of contaminants in this zone generally exceed those found anywhere else at or near the site (i.e., below the clay, in the Creek, or in ground water adjoining the site).

In selecting chemicals from among those detected at and near the site, factors that were considered included frequency of detection, concentrations detected, detection in more than one environmental medium, mobility potential, effects of the chemicals, and, for inorganics, presence in soil at above background levels. Table 2-14 summarized this list of chemicals of concern and the media in which they were detected at the site.

For each of these chemicals, health criteria (i.e., quantitative dose-response values) for both carcinogenic and noncarcinogenic effects associated with exposure were collected. The primary source of this information was the U.S. Environmental Protection Agency's (USEPA's) Integrated Risk Information System (IRIS) and Health Effects Assessment Summary Tables (HEASTs). The health criteria for carcinogenic effects are slope factors developed by USEPA's Carcinogen Assessment Group (CAG). The health criteria for noncarcinogenic

effects are reference doses (RfDs) generally developed by USEPA's RfD Work Group. The health criteria are generally derived using very conservative assumptions. As a result, potential risks predicted using these values are unlikely to underestimate actual risks although they may overestimate risks.

The following statements and conclusions can be made regarding the selected chemicals of concern:

(1) All are hazardous substances under CERCLA.

(2) None of the VOCs, pesticides, PCBs and semi-volatile compounds listed originate from natural sources but yet many of these chemicals exist at grossly elevated levels at the site.

(3) Some are possible human carcinogens (e.g., butyl benzyl phthalate, 1,1-dichloroethylene, isophorone, 1,1,2,2-tetrachloroethane).

(4) Many of the selected chemicals are probable human carcinogens (e.g., PCBs, chloroform, 1,2-dichloroethane, methylene chloride, trichloroethylene, cadmium [inhalation only]).

(5) Some are known carcinogens in humans (e.g. vinyl chloride, arsenic, benzene).

(6) Many exist in the water table aquifer at the site at levels which far exceed (often by orders of magnitude) the Maximum Contaminant Levels (MCLs) established for such substances pursuant to the Federal Safe Drinking Water Act (e.g., benzene and vinyl chloride [known human carcinogens], and chlorobenzene, chloroform, 1,2-dichloroethane, 1,2-trans-dichloroethylene, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane).

(7) Many exist in the till aquifer at the site at levels which exceed (often by orders of magnitude) the MCLs which were established for substances pursuant to the Federal Safe Drinking Water Act (e.g., 1,2-dichloroethane, trichloroethylene, 1,1-dichloroethylene, 1,1,1-trichloroethane, chlorobenzene, and vinyl chloride [a known human carcinogen]).

(8) Some chemicals exist in the bedrock aquifer at levels which exceed (in some cases by orders of magnitude) MCLs (e.g., 1,2-dichloroethane, trichloroethylene).

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(9) Some chemicals exist in the shallow water table aquifer at the site at levels which far exceed the Class GW-2 drinking water standards set by the State of New Jersey for such chemicals in this aquifer (e.g., PCBs).

(10) Many of the selected chemicals detected at the site are known to cause acute and/or chronic health effects (other than carcinogenic) in humans if ingested, inhaled, or dermally contacted in sufficient quantities.

(11) Many of the selected chemicals which exist at the site and which were also detected in the sediment of Peach Island Creek are known to be acutely and/or chronically toxic to aquatic organisms.

(12) Some of the selected chemicals which exist at the site and which were also detected in the sediment of Peach Island Creek are known to bioaccumulate and biomagnify in certain aquatic species (e.g., PCBs).

(13) Many of the selected chemicals are highly mobile in ground water (as indicated in Table 3 of the Dames and Moore [1990] RI).

(14) Almost all of the selected VOCs (14 of 17) which exist in the soil and ground water at the site were also detected in either the water column and/or sediment in Peach Island Creek.

(15) Almost all of the selected semi-volatile compounds (10 of 12) which exist in the soil and ground water at the site were also detected in the sediment in Peach Island Creek.

(16) Some of the selected chemicals which exist at the site were also detected in Peach Island Creek at levels which exceed the applicable standards for that creek (e.g., copper, mercury, nickel, zinc).

(17) The site is presently uncapped and open to the atmosphere. Many of the chemicals discovered at the site are known to be capable of volatilizing into the atmosphere and thereby migrating away from the site in ambient air.

(18) The site receives approximately seven million gallons per year of precipitation, some of which flows off the site in the form of surface runoff into Peach Island Creek. Some precipitation will also infiltrate into the shallow water table aquifer. No controls or catchment structures exist to prevent this migration at present. Therefore, many of the hazardous substances listed in Table E-1 may migrate into this creek, especially during and shortly after storm events with consequential unknown impacts on aquatic biota.

8.2 HUMAN EXPOSURE ASSESSMENT

Potential pathways by which human populations (workers, residents, etc.) could be exposed to chemicals at or originating from the site under current land use or hypothetical future land use conditions were identified and selected for evaluation. An important first step in identifying exposure pathways is to consider the mechanisms by which the chemicals of concern at the site may migrate in the environment.

8.2.1 POTENTIAL MIGRATION OF SITE-RELATED CONTAMINANTS

The potential migration routes for chemicals at the SCP site include:

(1) Migration from the soils into the ground water at the site;

(2) Migration from the shallow water table aquifer downward into the till aquifer;

(3) Migration from the till aquifer into the bedrock aquifer (which is presently used as a public water supply);

(4) Surface runoff from the site into Peach Island Creek;

(5) Migration of ground water directly into Peach Island Creek;

(6) Lateral migration of on-site ground water to off-site areas;

(7) Migration into the air by volatilization or particulate suspension; and

(8) Migration in Peach Island Creek of surface water and sediments.

An assessment of the potential environmental effects posed by some of these routes of migration was not possible given the limited data available at this

time (e.g., lateral migration into off-site ground water areas). Analysis of samples collected from soil, ground water, surface water, and sediment at the SCP site indicate, however, that chemicals of concern together with their transformation products are present in several or all of these media.

Many of the compounds detected in soil at the site will migrate due to the presence of infiltrating precipitation and the downward hydraulic gradient between the water table and till aquifers (Dames and Moore 1990). The substituted simple aromatics (e.g., chlorobenzene), chlorinated aliphatics (e.g., tetrachloroethylene) and phenols are expected to be more mobile than the other chemicals of concern in soil. The propensity of these chemicals to be mobile in soils is observed at the SCP site, where the substituted simple aromatics and chlorinated aliphatics have been observed in all soil depths sampled and in the water table, till, and bedrock aquifers. Based on the presence of several substituted simple aromatics and chlorinated aliphatics at elevated levels within the clay and in the underlying till aquifer, and at detectable levels in the bedrock aquifer, it can be concluded that hazardous substances are migrating into the bedrock aquifer.

The chlorinated pesticides, PAHs, PCBs, and phthalate esters detected in soils are generally not as mobile as the chemicals mentioned above. Many of these chemcials were, however, present at all the soil depths sampled at the SCP site. This is particularly true for PCBs and many of the PAHs indicating that downward transport is still occurring, although to a lesser extent than the

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simple substituted aromatics and chlorinated aliphatics. PCBs were also present in filtered water table aquifer samples at levels exceeding their solubility. This may result from the presence of organic solvents which may enhance the solubility (and thus mobility) of PCBs in ground water and in soil.

Many of the chlorinated aliphatics and substituted simple aromatics detected in Peach Island Creek adjacent to the site were also detected in the water table and till aquifers and in soils at the site (e.g., 1,1,1-trichloroethane, 1,2-trans-dichloroethylene). The similarity of compounds detected in Peach Island Creek as compared to those detected in the ground water and soil at the SCP site strongly suggest that chemical contaminants have migrated via ground water discharge and/or surface runoff from the site into the creek. However, given the limited surface water sampling program undertaken and the tidal nature of the creek, it is difficult to identify definitively the extent to which contaminants may be migrating into the creek from the site. There are, however, clear similarities between the types of chemicals detected in sediments and those detected on site in ground water and soil. These data further suggest that contaminants are migrating into Peach Island Creek from the site.

Chemicals present at the SCP site may migrate into the air in two ways, by volatilization or by suspension of soil (i.e., generation of fugitive dusts). Of the chemicals of concern at the SCP site, the chlorinated aliphatics (e.g.,

1,1-dichloroethane, 1,2-dichloroethane, 1,1-dichloroethylene, 1,2-trans-dichloroethylene, methylene chloride, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene, and vinyl chloride) and some of the substituted simple aromatics (e.g., benzene, 1,2-dichlorobenzene, toluene, and total xylenes) tend to volatilize readily from contaminated soils and water. While the other chemicals of concern such as the pesticides, phthalate esters, PAHs, and PCBs are less volatile, they still may be emitted from site soils into the air. Fugitive dust emissions could also occur at the SCP site in areas that are unpaved or unvegetated.

Biological and chemical processes that occur in the soil can also be important in determining the ultimate fate of organic chemicals found at the SCP site. These processes can, for example, produce more toxic and/or more mobile breakdown products. In most cases, an organic chemical occurring in the natural environment is not broken down immediately to carbon dioxide and water by a microorganism, but is metabolized to an intermediate which is in turn further degraded. These intermediates are typically more water soluble than the parent compound and are therefore more mobile. Many of the organic intermediates are also more toxic. For example, it is possible that the vinyl chloride in the water table and till aquifers at the SCP site occurs as a result of the transformation of the unsaturated higher molecular weight chlorinated aliphatics (e.g., trichloroethylene). Vinyl chloride is stable with respect to further biological and/or chemical transformation and is likely to persist unless it has an opportunity to volatilize or leach from

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soil. PCBs, which are comprised of mixtures of polychlorinated biphenyl congeners, may be metabolized by microorganisms present in the environment. Metabolism of one PCB congener will sequentially yield PCB congeners of lower molecular weight and greater solubility along with other metabolic byproducts such as PCB alcohols and/or ethers. The PAHs present in soil can also be biodegraded.

Most of the hydrophobic organics (e.g., PAHs, PCBs, dieldrin) and inorganics that may enter Peach Island Creek from the site tend to adsorb to organic and inorganic particulate matter in the waterway and subsequently deposit in the sediments. This contaminant burden usually remains relatively near the source, with concentrations decreasing approximately logarithmically with distance from the source. Many of the chemicals of concern in sediments in Peach Island Creek (e.g., trichloroethylene, toluene, Arochlor 1242, xylenes, and ethylbenzene) were in fact detected at their highest levels immediately adjacent to the site.

8.2.2 IDENTIFICATION OF EXPOSURE PATHWAYS

Based on a review of the site area and the results of the site investigation, a set of pathways through which humans may be exposed currently or in the future to site-related contaminants was identified for detailed evaluation. The exposure pathways that were evaluated for both current and future site and nearby land use conditions were as follows:

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Current Site and Nearby Land Use Conditions

- Direct Contact with Site Surface Soil by Trespassers
- Inhalation of Volatilized Organics by Nearby Residents and Workers
- Inhalation of Suspended Soil by Nearby Residents and Workers

Future Site and Nearby Land Use Conditions

- Direct Contact with Surface Soil by Future On-Site Workers
- Inhalation of Volatilized Organics by Future On-Site Workers
- Inhalation of Suspended Soil by Future On-Site Workers
- Ingestion of Ground Water by Future On-Site Workers
- Direct Contact with Subsurface Soil by Future Construction Workers

Other potential pathways of exposure which may exist but which were not evaluated in the assessment (e.g., due to insufficient sampling data) include: incidental ingestion of surface water and sediments from Peach Island Creek, ingestion of fish or shellfish from Peach Island Creek, and ingestion of any ground water in the vicinity of the site. In addition, exposures to chemicals in ground water through routes other than ingestion (e.g., inhalation of volatiles released from water into indoor air) were not quantitatively evaluated.

In order to evaluate exposures for each pathway, scenarios were developed based on estimates regarding the extent, frequency, and duration of exposures. In addition, the concentrations to which individuals might be exposed were calculated based on the site sampling data except for the inhalation pathways for which emission and dispersion models were used to estimate air concentrations. These concentrations are referred to as exposure point concentrations.

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For each exposure pathway, the potential exposure to individuals was estimated for both an average case and, in accordance with recent USEPA guidance (USEPA 1989a) at the request of USEPA Region II, a reasonable maximum exposure (RME) case. The average case combines average exposure point concentrations with average estimates for the extent, frequency, and duration of exposure. The average case is designed to be roughly representative of exposures a "typical" individual might experience. For the same receptor locations, the RME case combines the maximum exposure point concentrations with RME values, described and in many cases specifically provided by USEPA (1989a), describing the extent, frequency, and duration of exposure. The RME scenario incorporates the exposure parameter values recommended in USEPA's (1989a) Superfund guidance to the extent possible given that this project was well underway at the time the new USEPA (1989a) guidance was released. The RME case is designed to represent an upper bound on potential exposures; that is, predicted exposures are likely to overestimate actual risks but are unlikely to underestimate actual risks.

8.3 HUMAN RISK CHARACTERIZATION

Risks from the above exposures were evaluated first by comparing concentrations of chemicals in the contaminated exposure medium (e.g., ground water) at point of potential exposure, to State or Federal environmental standards, criteria, or guidance that were identified as "Applicable or

Relevant and Appropriate Requirements" (ARARs) or other relevant guidance. In addition, exposures were also evaluated by quantitative risk assessment.

The soil sampling data from the site indicate that numerous chemicals exceeded the NJDEP soil cleanup objectives as shown in Table 8-1. These include total volatile organics, total base neutral and acid extractable compounds, PCBs, arsenic, chromium, cadmium, lead, mercury, and zinc.

A wide variety of chemicals also exceeded federal and state standards and guidelines for ground water. The ARARs and other guidance that were used in this comparison were federal maximum contaminant levels (MCLs) and MCL goals (MCLGs), federal ambient water quality criteria for protection of human health adjusted for drinking water exposures only, state MCLs, and state ground water standards. The chemicals which exceeded several of these ARARs and other guidance levels included benzene, chlorobenzene, 1,2-dichloroethane, trichloroethylene, chloroform, and many other VOCs, PCBs, total PAHs, phenol, arsenic, cadmium, chromium, lead, mercury and nickel.

For the quantitative assessment of risks, exposure estimates were combined with the health criteria for the selected chemicals of concern to estimate potential risks to human health. As for exposures, risks are estimated for an average and a RME case. The average case combines the average case exposure estimates with generally upper bound slope factors and conservatively derived reference doses. This average case is intended to represent the exposure of a

typical individual; however, use of conservative health criteria may result in an overestimation of risk even for the average case. The RME case combines the RME exposure estimates with generally upper bound slope factors and conservative reference doses. This scenario is intended to place a conservative upper bound on the potential risks.

It should also be kept in mind that the risks reported in this BRA are estimates of current or potential risks to human health under the average or RME exposure pathways evaluated.

Table 8-1 summarizes the risk estimates for the exposure pathways evaluated in this BRA. In this table, the pathways for which the total (i.e., summed across chemicals within a pathway) potential upper bound lifetime excess cancer risks exceeded $1x10^{-6}$ (one in one million) are identified. An upper bound excess lifetime cancer risk of $1x10^{-6}$ means that an individual's incremental chance of developing cancer over a 70-year lifetime due to the specific exposure conditions evaluated is one in one million at most. The USEPA's target risk range for evaluating Superfund sites is from $1x10^{-6}$ (one in one million) to $1x10^{-4}$ (one in ten thousand). Also listed in this table are those individual chemicals for which the upper bound excess lifetime cancer risks exceeded $1x10^{-6}$. The exposure pathways for which total excess lifetime cancer risks across chemicals exceeded $1x10^{-6}$ were as follows:

(1) Trespassers who may contact on-site surface soil (average and reasonable maximum cases). The risks were predominantly due to exposure to aldrin, arsenic, carcinogenic PAHs, 1,1-dichloroethane, dieldrin, PCBs, tetrachloroethylene, and trichloroethylene.

(2) Possible future on-site workers who may regularly contact surface soil
    (average and reasonable maximum cases). The risks were predominantly due
    to exposure to aldrin, arsenic, benzene, bis(2-ethylhexyl)phthalate,
    carcinogenic PAHs, tetrachloroethylene, 1,1-dichloroethane, 1,2-
    dichloroethane, dieldrin, PCBs, tetrachloroethylene, and
    trichloroethylene.

(3) Possible future on-site construction workers who may contact subsurface
    soil (reasonable maximum case only). The risks were predominantly due to
    exposure to carcinogenic PAHs and PCBs.

(4) Possible future on-site workers who may regularly consume ground water
    from the on-site water table, till, and bedrock aquifers. It should be
    noted that, for the water table and till aquifers, this pathway is
    unlikely to occur since these aquifers are not known to be used for water
    supply in the area. These aquifers were evaluated, however, because of
    the likelihood of migration from these aquifers to the bedrock aquifer
    which is used for drinking water in the area. The risks from use of the
    water table and till aquifer ground water for potable uses were associated
    with exposure to numerous volatile organic compounds, carcinogenic PAHs,
    and PCBs. The risks from use of the bedrock aquifer ground water were
    associated with exposure to volatile organic compounds.

(5) Workers currently employed near the site who may inhale volatilized
    organics and suspended soil released into the air (reasonable maximum case
    only). The risks were predominantly due to exposure to chromium, 1,1-
    dichloroethylene, and vinyl chloride.

(6) Possible future on-site workers who may inhale volatilized chemicals or
    suspended on-site soil. The risks were predominantly due to chloroform,
    chromium, 1,1-dichloroethylene, methylene chloride, trichloroethylene, and
    vinyl chloride.

Table 8-1 also indicates which exposure pathways may result in adverse

noncarcinogenic effects. For the following pathways, there is a potential for

such adverse effects to occur: (1) Trespassers who may contact on-site

surface soil (maximum case only) and (2) Possible future on-site workers who

may regularly contact surface soil (maximum case only). These risks were

predominantly due to exposure to aldrin and lead. (3) Possible future on-site

workers who may regularly consume on-site ground water from the water table

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and till aquifers (although this pathway is unlikely to occur). These risks were predominantly due to numerous volatile organic compounds, nitrobenzene, and lead.

8.4 ECOLOGICAL RISK ASSESSMENT

Potential risks to environmental receptors were also evaluated for the SCP site using the results of site monitoring data, a review of the toxicity of the chemicals of concern, and estimates of exposure. Risks have been characterized by comparing chemical concentrations with federal criteria and by comparing estimated exposures with toxicity values provided in the scientific literature.

The potential risks from exposures of aquatic and terrestrial wildlife to chemicals in surface water and sediments of Peach Island Creek were assessed. The site is not currently a habitat for terrestrial wildlife or plants (with the exception of some sparse vegetation). Thus, since these ecological exposure pathways are not currently considered complete, they were not evaluated in this assessment. However, the absence of terrestrial wildlife and abundant plant life may be due, in part, to contamination at the site. The results of the environmental risk assessment are summarized below.

The potential risks to aquatic life were evaluated by comparing surface water and sediment chemical concentrations with USEPA ambient water quality criteria (AWQC), sediment quality criteria (SQC), and other toxicity values. It was

concluded that risks to aquatic life may occur from exposure to chemicals in surface water since ambient water quality criteria were exceeded for copper, mercury, nickel, and zinc. Risks to aquatic life from chemicals in sediments are also likely to occur since: (1) dieldrin and PCB concentrations exceed sediment quality criteria, (2) copper concentrations in sediments exceed concentrations that were shown to be lethal in short-term laboratory tests with invertebrates, and (3) concentrations of cadmium, lead, mercury and zinc exceed levels that have produced biological effects in sediment toxicity tests conducted in west coast estuaries. In addition, PCB concentrations in sediments exceed the proposed 0.1 mg/kg preliminary action level by factors of 52 to 550. These comparisons imply that populations of aquatic life that may inhabit Peach Island Creek may be at risk. Thus it is likely that adverse effects are occurring from exposure of aquatic life to contaminants in surface water and sediments.

Additionally, potential risks to the Pied-billed Grebe, which has an endangered breeding population in the Meadowlands not far from Berry's Creek, were evaluated. Estimated dietary concentrations (from consumption of invertebrates in which sediment contaminants have bioaccumulated) of dieldrin, cadmium, copper, lead, mercury, nickel, and zinc exceed toxicity values derived from toxicological studies. There are considerable uncertainties associated with these estimated dietary concentrations since they are based on: (1) limited sediment sampling data, (2) estimation of sediment pore water concentrations by multiplying sediment concentrations by a sediment:water partition coefficient, and (3) use of short-term invertebrate bioconcentration

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factors. These data indicate, however, that adverse effects to this endangered breeding population might occur should the population exist near the site or feed extensively from Peach Island Creek. Birds that frequent other, less contaminated areas for a portion of their diet, would be at less risk.

It is unlikely that mammalian wildlife such as the muskrat will suffer adverse effects from the ingestion of contaminants in surface water. Estimated doses were at least one order of magnitude below toxicity values for all chemicals of concern. Other possible routes of exposure for mammalian wildlife, such as through the diet or contact with sediments, have not been quantified because currently available data are insufficient to estimate doses or dietary levels.

8.5 CONCLUSIONS

Based on the results of this Baseline Risk Assessment it can be concluded that the SCP site has caused severe degradation of the water table and till aquifers at the site. The concentrations of numerous chemicals in the water table and till aquifers exceed both federal and state drinking water standards and guidelines. Contaminants at the site have also migrated into the bedrock aquifer which is used as a public drinking water supply source. Results of preliminary testing from the bedrock aquifer indicate that certain chemicals have already migrated from the site into this drinking water source. In fact, concentrations of several site-related chemicals in the bedrock aquifer exceed both federal and state drinking water standards and guidelines. In addition,

contaminants from the site are migrating into Peach Island Creek. The concentrations of several chemicals in surface water and sediments of this creek exceed levels associated with adverse effects in aquatic life, although it is not possible (at present) to quantify the site-related impact. Soil contact by site trespassers and future on-site workers would result in adverse human health impacts under the exposure scenarios evaluated. Furthermore, the concentrations of several chemicals in on-site soils exceed available state and federal guidelines.

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SECTION 6

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APPENDIX A

CHEMICAL AND PHYSICAL PARAMETERS FOR ORGANIC CHEMICALS
DETECTED AT THE SCP SITE

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CHEMICAL AND PHYSICAL PARAMETERS FOR ORGANIC CHEMICALS DETECTED
AT THE SCP SITE

Chemical	Vapor Pressure mm Hg @ 20°C	Water Solubility mg/l @ 20°C	Octanol/Water Partition Coefficient (log₁₀)	Soil/Sediment Adsorption Coefficient (log₁₀)	Specific Gravity (20°C)
Volatile Compounds					
Benzene	76(1)	1780(1)	2.13(3)	1.99(5)	0.879(1)
Chlorobenzene	8.8(1)	500(1)	2.84(3)	2.25(7)	1.1066(1)
Chloroethane	1000(1)	5740(1)	1.54(3)	1.57(7)	0.92(1)
Chloroform	160(1)	8000(1)	1.97(3)	1.59(7)	1.489(1)
1,1-Dichloroethane	180(1)	5500(1)	1.79(3)	1.63(7)	1.174(1)
1,2-Dichloroethane	61(1)	8690(1)	1.48(3)	1.52(7)	1.25(1)
1,1-Dichloroethylene	500(1)	400(1)	1.48(2)	2.26(7)	1.218(1)
Ethyl benzene	7(1)	152(1)	3.15(3)	2.95(5)	0.867(1)
Methylene chloride	349(1)	20000(1)	1.25(3)	1.16(7)	1.326(2)
1,1,2-2-Tetrachloroethane	5(1)	2900(1)	2.56(3)	1.92(7)	1.60(1)
Tetrachloroethylene	14(1)	150(1)	2.88(3)	2.63(7)	1.626(1)
Toluene	22(1)	515(1)	2.69(3)	2.51(5)	0.867(1)
1,2-Trans-dichloroethylene	200(1)	600(1)	1.48(3)	2.17(7)	1.26(1)
1,1,1-Trichloroethane	100(1)	4400(1)	2.17(3)	1.75(7)	1.35(1)
1,1,2-Trichloroethane	19(1)	4500(1)	2.17(3)	1.75(7)	1.44(1)
Trichloroethylene	60(1)	1,100(1)	2.29(3)	3.76(7)	1.46(1)
Vinyl chloride	2660(1) @ 25°C	1,100(1) @ 25°C	0.60(3)	1.91(7)	0.912(1)
Methyl ethyl ketone	77.5(1)	3.53 x 10⁵(1)	0.26(1)	0.59(6)	0.805(1)
Styrene	5(1)	300(1)	2.69(14)	2.62(9)	0.905(1)
Total Xylenes	6(1)	180(1)	3.02(1)	2.84(7)	0.864(1)
Acid Compounds					
2-Chlorophenol	5 @ 72°C(1)	26000(1)	8.52(3)	1.32(7)	1.245 @ 45°C(1)
2,4-Dichlorophenol	0.12(3)	4600(1)	2.75(3)	1.80(7)	1.383(1)
2,4-Dimethylphenol	0.06(3)	17000(3)	2.50(3)	2.34(5)	1.036(1)
2-Nitrophenol	20 @ 105°C(1)	2100(1)	1.76(3)	1.64(5)	1.657(1)
Phenol	0.2(1)	82000(1)	1.46(3)	1.36(5)	1.07(1)
Base/Neutral Compounds					
Acenaphthene	10⁻³ - 10⁻²(3)	3.4(3)	4.33(3)	4.05(5)	1.024(4)
Acenaphthylene	10⁻³ - 10⁻²(3)	3.9(3)	4.07(3)	3.81(5)	0.899(4)
Anthracene	2 x 10⁻³(3)	0.075(1)	4.45(3)	4.16(5)	1.24(1)
Benzidine	5 x 10⁻¹⁴(11)	400 @ 12°C(1)	1.81(3)	1.69(5)	1.25(1)
Benzo(a)anthracene	5 x 10⁻⁹(3)	0.01(3)	5.61(3)	5.25(5)	NA
Benzo(a)pyrene	5 x 10⁻⁹(3)	0.004(3)	6.04(3)	5.65(5)	NA
Benzo(b)fluoranthene	10⁻¹¹ - 10⁻⁶(3)	0.009(12)	6.57(3)	6.15(5)	NA
Benzo(ghi)perylene	10⁻¹⁰(3)	3 x 10⁻⁴(3)	7.23(3)	6.77(5)	NA
Benzo(k)fluoranthene	9.6 x 10⁻¹¹(3)	0.0016(12)	6.84(3)	6.40(5)	NA
bis(2-Chloroethyl)ether	0.7(3)	10200(3)	1.58(3)	1.47(5)	1.220(4)
bis(2-Ethylhexyl)phthalate	2 x 10⁻⁷(3)	0.4(3)	8.73(3)	8.17(5)	0.99(1)

Chemical	Vapor Pressure mm Hg @ 20°C	Water Solubility mg/l @ 20°C	Octanol/Water Partition Coefficient ($\log_{10}$)	Soil/Sediment Adsorption Coefficient ($\log_{10}$)	Specific Gravity (20°C)
Base/Neutral Compounds					
Butyl benzyl phthalate	8.6×10^{-6}(1)	2.9(3)	5.8(3)	5.43(5)	1.1(1)
2-Chloronaphthalene	0.02(3)	6.7(3)	4.12(3)	NA	1.138(4)
Chrysene	$10^{-11} - 10^{-6}$(3)	0.002(3)	5.61(3)	5.25(5)	1.274(4)
Dibenzo(a,h)anthracene	10^{-10}(3)	5×10^{-4}(3)	5.97(3)	5.59(5)	NA
1,2-Dichlorobenzene	1.5(3)	100(1)	3.38(3)	2.70(7)	1.304(1)
1,3-Dichlorobenzene	2.3(3)	123(1)	3.38(3)	2.65(7)	1.288(1)
1,4-Dichlorobenzene	0.6(1)	79(1)	3.39(3)	2.76(7)	1.458(1)
Diethyl phthalate	0.05(3)	1000(3)	3.22(3)	3.01(5)	1.12(1)
Di-n-butyl phthalate	0.1(3)	13(3)	5.2(3)	4.87(5)	1.0465(1)
Di-n-octyl phthalate	0.2(3)	3(3)	9.2(3)	8.61(5)	0.99(1)
Fluoranthene	$10^{-6} - 10^{-4}$(3)	0.3(3)	5.33(3)	4.99(5)	1.252(4)
Fluorene	$10^{-3} - 10^{-2}$(3)	2(3)	4.18(3)	3.91(5)	1.203(4)
Indeno (1,2,3-c,d)pyrene	10^{-10}(3)	0.0034 @ 25°C(2)	7.66(3)	7.17(5)	NA
Isophorone	0.4(1)	12000(1)	1.7(3)	1.59(5)	0.92(1)
Naphthalene	0.5(3)	30(1)	3.37(3)	3.15(5)	1.152(1)
Nitrobenzene	0.15(1)	1,900(1) (8,000 @ 80°C)(1)	1.85(3)	1.73(5)	1.20(1)
N-Nitrosodiphenylamine	0.007(11)	1100(12)	2.57(3)	2.40(5)	NA
Phenanthrene	6.8×10^{-4}(3)	1.6(1)	4.46(3)	4.17(5)	1.025(1)
Pyrene	6.9×10^{-7}(3)	0.1(3)	5.32(3)	4.98(5)	1.271(1)
1,2,4-Trichlorobenzene	0.4(3)	30(3)	4.26(3)	3.04(7)	1.574(1)
Pesticide Compounds					
Aldrin	7.5×10^{-5}(2)	0.017(3)	NA	4.61(6)	NA
Beta-BHC	NA	NA	NA	NA	NA
4,4'-DDT	1.5×10^{-7}(2)	0.006(3)	6.19(3)	4.86(6)	NA
4,4'-DDE	6.5×10^{-6}(3)	0.04(3)	5.69(3)	4.41(6)	NA
Dieldrin	3.1×10^{-6}(2)	0.2(3)	5.6(3)	4.02(6)	1.75(1)
Endosulfan I	1×10^{-5}(3)	0.5(3)	3.60(3)	3.81(6)	NA
Endrin	2×10^{-7}(2)	0.3(3)	5.6(3)	3.93(6)	NA
Endrin aldehyde	2×10^{-7}(3)	0.3(3)	5.6(3)	3.93(6)	NA
Methoxychlor	NA	NA	NA	NA	NA
PCB-Aroclors					
Aroclor 1242	4.1×10^{-4}(3)	0.34(3)	4.11(3)	3.85(5)	1.35(3)
Aroclor 1254	7.7×10^{-5}(3)	0.056(3)	6.03(3)	5.64(5)	1.50(3)
Aroclor 1260	4.1×10^{-5}(3)	0.0027(3)	7.14(3)	6.68(5)	1.58(3)
Aroclor 1248	4.9×10^{-4}(3)	0.054(3)	5.75(3)	5.38(5)	1.41(3)
Aroclor 1232	4.1×10^{-3}(3)	1.5(3)	3.2(3)	2.99(5)	1.24(3)

NOTES:

1. Reference 12

2. Reference 11

3. Reference 13

4. Reference 14

5. Reference 10 – Equation 4.9: $\log K_{oc} = 0.937 \log K_{ow} - 0.006$

6. Reference 10 – Equation 4.5: $\log K_{oc} = -0.55 \log S + 3.64$ (S = water solubility in mg/1)

7. Reference 10 – Equation 4.7: $\log K_{oc} = -0.557 \log S + 4.277$ (S in u moles/1)

8. Average values for ortho, meta and para xylene

9. Average values for equations 4.6 ($\log K_{oc} = -0.54 \log S + 0.44$, S is mole fraction) and 4.10 ($\log K_{oc} = 1.00 \log K_{ow} - 0.21$)

10. Reference 10 – Equation 2.3

11. Reference 10 – Equation 14.20

12. Reference 10 – Equation 2.20

13. Reference 15

14. Reference 10 – Chapter 1

NA. Not Available

NC. Not Calculated

Source: Dames and Moore (1988)

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APPENDIX B

MODELS USED TO CALCULATE AIR CONCENTRATIONS FOR THE SCP SITE

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SOIL VOLATILIZATION EMISSIONS MODEL

Mathematical models have been developed to predict volatilization rates of organic chemicals from the soil when the chemical concentration in the soil is known. One such model proposed by Hwang (1986) was used to calculate the emissions of volatile organic compounds due to soil volatilization at the Carlstadt site. The Hwang model used in this assessment calculates the flux as a function of time for chemicals incorporated throughout the soil column from the soil surface. A time dependent model was used because the organic chemicals in the soils at the Carlstadt site represent a finite reservoir of contamination. As organic chemicals volatilize from the Carlstadt site the concentration in the soil will decrease. Because the volatilization flux rate is proportional to the chemical concentration in the soil, the reduction in soil concentration will result in a decreased volatilization flux rate. Additionally, it is possible that the initial mass of a particular chemical could be totally depleted from the source in less than a 70-year exposure period, so that calculating volatilization for a 70-year period could result in unrealistically high risks.

To account for the reduced volatilization flux rate due to source depletion, the Hwang model was run for a one-year period (for volatilization modeling, eight months was assumed to equal one year because for four months of the year the soil would be frozen which would probably halt the volatilization process). The mass lost over the year was subtracted from the mass of the chemical in the soil at the beginning of that year, and a new soil concentration was calculated. The new soil concentration was used in the Hwang model to calculate the volatilization flux for the next year. This process was continued until the volatilization flux was reduced by a factor of ten million (10^7) from the initial flux or until the reservoir was totally depleted for a given organic chemical.

An average flux rate for the exposure period was calculated by summing the flux rates for each year and dividing by the number of years over which the 10^7 reduction in volatilization flux occurred. Typical values of

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averaging periods obtained using this method for the selected volatile chemicals of concern were 20-30 years. It should be noted that the average flux obtained here may be as much as an order of magnitude higher than if the exposure period was assumed to be over a lifetime of 70 years. The average flux rate was then used in the air dispersion models to determine on-site and off-site air concentrations associated with the volatilization of organic chemicals from the soil.

The time-dependent volatilization model used in this exposure scenario assumes that the chemicals are incorporated uniformly throughout the soil column. In the soil matrix a chemical can exist in the following three phases: adsorbed to soil particles; as a liquid in the soil pore spaces; or as a vapor also in the soil pore spaces. In the Hwang model the flux rate of chemicals from the soil into the air is a result of Fickian diffusion of chemical vapors up through the soil matrix. Thus it was necessary to determine the concentration of vapors in the soil pore spaces associated with the reported concentrations of chemicals adsorbed to soil particles at the Carlstadt site. By assuming an equilibrium partitioning between the adsorbed, liquid and gas phases of a chemical in the soil matrix, it was possible to determine the chemical concentration in each phase.

The phase partitioning between the adsorbed and liquid phases is a function of the fraction of organic carbon (f_{oc}) in the soil and the tendency for the compound to be adsorbed by the organic matter. The value for f_{oc} for soils at the Carlstadt site was assumed to be 1.0%. The tendency for a chemical to be adsorbed to organic matter in the soil can be described by the organic carbon partition coefficient (K_{oc}). The equilibrium concentration of a chemical in solution was determined by:

$$C_1 = \frac{C_s}{K_{oc} f_{oc}}$$

where

C_1 = concentration of chemical in solution, [g/ml]

C_s = concentration of chemical adsorbed to soil, [g/g].

The concentration of a chemical in solution was used with the liquid-vapor partition coefficient to determine the equilibrium vapor-phase concentration. The liquid-vapor partition coefficient is generally represented by the chemical-specific Henry's Law Constant. Thus the vapor-phase concentration of a chemical in the soil pore spaces was determined by:

$$C_g = \frac{C_1 H}{R T}$$

where

C_g = concentration of chemical in the vapor phase, $[g/cm^3]$

H = Henry's Law Constant, $[atm\text{-}m^3/mol]$,

R = universal gas constant, $[8.19 \times 10^{-5} \; atm\text{-}m^3/mol\text{-}K]$

T = soil temperature, $[293 \; K]$.

The soil column was assumed to be isothermal with a constant temperature of 20°C. This assumption will yield an average estimate of the annual volatilization flux since periods of lower soil temperatures at the Carlstadt area site could retard or even halt the volatilization process, while higher temperatures could accelerate the process.

Based on the assumption of equilibrium phase partitioning in the soil matrix, the vapor phase concentration of a chemical C_g in the Hwang time-dependent flux rate equation is given by:

$$C_g = \frac{H}{R T} C_1 = \frac{H}{R T} \frac{1}{K_d} C_s = \frac{H'}{K_d} C_{so} = \frac{H'}{K_{oc} f_{oc}} C_{so}$$

where C_{so} is the initial concentration of chemical adsorbed to the soil and all other variables are as defined earlier.

The transport of chemical vapors in the soil is by diffusion through the soil pore spaces. The effect of soil geometry and moisture on the vapor-phase diffusion is accounted for by defining an effective diffusivity, D_s. The effective diffusivity is given by:

$$D_s = D_i * E^{1/3}$$

where

  D_i = vapor-phase diffusion coefficient in air for the chemical, cm^2/s

  E = soil porosity.

This definition for the effective diffusivity assumes that the soil is dry.

  The flux rate of vapors from the soil surface into the overlying air is obtained by solving a mass-balance equation for a vertical element of the soil. Solution of this equation requires the specification of initial and boundary conditions. In this case the assumed conditions are a time dependent emission from the soil surface, with chemicals incorporated throughout the soil column, beginning at the soil surface. The initial condition is that the vapor-phase concentration throughout the soil pore spaces is given by the equilibrium partitioning described earlier. The boundary condition for the soil surface sets the vapor-phase concentration equal to zero. The lower boundary condition sets the vapor-phase concentration equal to the equilibrium concentration to an infinite depth. Using these initial and boundary conditions, the average flux rate for a given time period, t, is given by:

$$N_a = \frac{2\ E\ Ds}{\sqrt{\pi\ \alpha\ t}}\ \frac{H'}{K_d}\ C_{so}$$

where

  N_a = average flux rate over the period t, $[g/m^2\text{-}s]$

  E = total porosity

  Ds = effective diffusivity, $[cm^2/s]$

  H' = nondimensional Henry's Law constant

  K_d = soil/liquid partition coefficient = Koc * foc, $[cm^3/g]$

  C_{so} = initial chemical concentration in the soil, $[g/g]$

  t = flux rate period, s

  α = $[D_s * E]/[E + P_s * (1 - E)(K_d/H')]$, $[cm^2/s]$

    where P_s = true soil density, $[g/cm^3]$.

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The total porosity was set at 0.52. The chemical-specific value for K_d was the product of the chemical-specific K_{oc} value and the soil f_{oc} value, assumed to be 1.0%. The soil was assumed to maintain a constant 20°C temperature. The flux rate period was 20,736,000 seconds (8 months). The true soil density was computed as defined by Hwang (1986) as the ratio of the soil bulk density and the total soil porosity. The soil bulk density was assumed to be 1.33 g/cm^3. For chemical classes, such as PAHs and PCBs, chemical-specific parameters for one member of the class (the member for which a dose-response value was available) were used to estimate emissions. These chemicals were benzo(a)pyrene for carcinogenic PAHs, naphthalene for noncarcinogenic PAHs, and Aroclor 1254 for PCBs.

FUGITIVE EMISSIONS FROM WIND EROSION

Contaminants were found in the surface soils of the Carlstadt site. Since soil surfaces at the site are not completely covered by vegetation, wind entrainment of dust particles is a potential pathway for inhalation of contaminants.

Airborne particulate matter with an aerodynamic diameter less than or equal to 10 um, referred to as PM_{10}, is respirable and, when contaminated, can contribute to inhalation exposure (Cowherd et al. 1984). The methodologies described by Cowherd et al. (1984) were used to calculate the emission rates of PM_{10}. Emission rates were determined from emission factors, the source extent, and the mass fraction of the chemicals in the soil. The PM_{10} emission factors employed in this method were empirically derived through regression analysis of field test data (Cowherd et al. 1984). The calculated PM_{10} emission rates were then linked to atmospheric dispersion models which predicted the ambient air concentrations of respirable particle matter on- and off-site.

The first step in estimating PM_{10} emissions associated with wind erosion of unvegetated portions of the contaminated site is the classification of the soil surface material. The soil surface is classified as having either a

"limited reservoir" or an "unlimited reservoir" of erodible surface particles.
Different equations are used to determine the wind erosion from these two
classes of soil surfaces.

The Carlstadt site contains no natural surface soils. The site consists
of construction debris and fill material which was imported to the site. Some
of the materials identified at the site were shingles, wood, brick, crushed
stone, red shale blocks, sand and gravel. The materials ranged in size from
less than 1 inch to over 6 inches. The average depth of this fill material
was approximately 8.4 feet (Dames and Moore Remedial Investigation 1988). Due
to the nature of the surface materials, it was assumed that the site would
have a "limited" erosion potential.

Consequently, the following equation from Cowherd et al. (1984) was
applied to determine particulate emissions from surfaces with a "limited"
reservoir of erodible material:

$$E_{10} = \frac{0.83(F)P(u^+)(1-V)}{(PE/50)^2}$$

where

E_{10} = PM$_{10}$ emission factor, i.e., annual average PM$_{10}$ emission rate per unit area of contaminated surface (mg/m^2-hr),

F = Frequency of disturbance per month,

u^+ = Observed (or probable) fastest mile or wind for the period between disturbances (m/s),

$P(u^+)$ = Erosion potential, i.e., quantity of erodible particles present on the surface prior to the onset of wind erosion (g/m^2),

V = Fraction of contaminated surface area covered by continuous vegetative cover (equals 0 for bare soil), and

PE = Thornthwaite's Precipitation Evaporation (PE) Index used as a measure of average soil moisture content.

Based on meteorological data from the Newark International Airport, the
annual percentage of time that the wind exceeds 6.8 m/s is 31%. Assuming that
this percentage is valid for monthly disturbances, then the frequency of

disturbances per month is 9.3 mo^{-1}. (This assumes that a wind speed greater than 6.8 m/s will cause a disturbance in the soil surface and uncover new erodible material.)

The erosion potential $P(u^+)$ is based on the following equation from Cowherd et al. (1984):

$$P(u^+) = 6.7 \; (u^+ - u_t)$$

Where u^+ is the annual average fastest mile and u_t is the erosion threshold wind speed at a typical weather station sensor height of 7 m. The annual average fastest mile (u^+) was determined to be 22.95 m/s from local climate data for Newark, NJ.

In order to obtain u_t, it was first necessary to determine a threshold friction velocity at the surface (u_{*t}). Entrainment of particle matter is dependent on wind speed. For a given soil type there is a surface threshold wind velocity which must be attained to initiate entrainment. Based on Cowherd et al. (1984) the dividing line between an "unlimited" and "limited" erosion potential is a surface friction velocity of 75 cm/s. Surfaces with friction velocities greater than 75 cm/s often are composed of aggregates too large to be eroded. For the Carlstadt assessment a value of 75 cm/s for the surface threshold friction velocity was used as this represents the most conservative value for a "limited" erosion surface. The value of u_t can be derived from u_{*t} using Figure 4-1 in Cowherd et al. (1984). The value of u_t was determined to be 8.24 m/s. The value of u_t and the annual average fastest mile u^+ were used in the preceding equation and an erosion potential of 98.5 g/m^2 was calculated.

Based on photographs of the site the fraction of vegetation, V, is assumed to be 50%. According to Cowherd et al. (1984) Figure 4-2, the PE Index for New Jersey is 120. The input parameters for the emission rate equation and the calculated emission rate are listed in Table 1.

TABLE 1

INPUT PARAMETER FOR THE "LIMITED" WIND EROSION MODEL

$$f = 9.3 \text{ mo}^{-1}$$

$$u^t = 22.95 \text{ m/s}$$

$$P(u^t) = 98.5 \text{ g/m}^2$$

$$V = 50\%$$

$$PE = 120$$

$$E_{10} = 1.83 \times 10^{-5} \text{ g/m}^2\text{-s}$$

To obtain the annual chemical-specific PM_{10} emission rate due to wind erosion, R_{10}, the PM_{10} emission factor, E_{10w}, is multiplied by the weight fraction of the chemical measured in the soil:

$$R_{10} = (W)(E_{10w})(2.78 \times 10^{-4} \text{ hr/sec})$$

where

R_{10} = emission flux of contaminant $(g/m^2/sec)$,

E_{10w} = annual PM_{10} emission rate due to wind erosion $(g/m^2/hr)$, and

W = mass fraction of chemical in surface soils (g/g).

The mass fraction for each chemical used in the PM_{10} emission rate equation was obtained by converting the reported chemical concentrations in the soil (in mg/kg) to a unitless value of g/g by using the appropriate conversion multipliers. It was assumed that the concentration in the PM_{10} particles was equal to the chemical concentrations measured in the bulk soil. The predicted chemical emission rates were then used to estimate the chemical-specific ambient concentrations on- and off-site by linking them to the appropriate air dispersion model. Off-site ambient air concentrations were predicted using the ISCLT model and on-site ambient air concentrations were predicted using the Box model. Both air dispersion models will be described in following sections of this appendix.

BOX MODEL

The ISCLT model cannot be used to determine the ambient concentrations of an area source at or near the source of emission. For this study, a box model was used to determine the ambient contaminant concentrations for future potential workers who would work at the Carlstadt site.

The box model assumes steady and spatially uniform conditions of dispersion so that the emissions from an area source are uniformly distributed

throughout a box defined by the area of the source and the mixing height. The model requires steady-state emission rates, a constant wind vector, and also that the crosswind distance of the area source is large in comparison to the downwind distance of the receptor. To meet these requirements, all emission rates were calculated for steady state, the wind speed was chosen to be the annual average wind speed recorded at a local airport, and the receptor location was the site of the area source. The only condition left to determine was the height of the box. Box models used on an urban scale often use the height of the daytime mixing layer, 500 m, as the height of the box. For that definition to be appropriate, a downstream fetch on the order of tens of kilometers is required. The mean vertical displacement of emissions as a function of stability and downwind distance should provide a reasonable analogy to the mixing height used in larger scale box models. The height of the box was determined using the following equation presented by Pasquill (1975):

$$X = 6.25 \ Z_0 \ [(H/Z_0) \ \ln \ (H/Z_0) - 1.58(H/Z_0) + 1.58))]$$

This expression is for a D or neutral stability class and should provide an average estimate of ambient concentrations because the effect of a change in atmospheric stability would be to raise or lower the box height relative to the neutral stability. Changes in the box height will affect the ambient concentrations in the box since the volume available for diluting the emissions is changed. The value for Z_0, the roughness height, was chosen to be 0.1 m which represents terrain with low crops and occasional large obstacles (NOAA 1983). The downwind distance, X, was chosen to be 168 m, which is the square root of the area of emissions at the Carlstadt site. This value is an approximation of the length of one side of the site and assumes that the site is square. Because the actual site is rectangular, ambient concentrations determined by the box model would be lowest when the wind is blowing parallel to the longest side of the site and highest when the wind is blowing parallel to the shortest side of the site, for the same conditions.

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Thus, treating the site as a square should provide an average case for the ambient concentrations.

Having specified X and Z_0, H was determined to be 8.8 m. This height represents the mean vertical height that a particle would attain after traveling across the entire length of the site. Because exposure to emissions could occur anywhere on the site and not just at the downwind edge, an average annual concentration was determined by using one-half the calculated value for the box height in the box model equation.

The concentration on-site can be determined using the equation:

$$C_i = \frac{Q_{vi}\ A}{(H/2)WU}$$

where

C_i = The concentration on-site for the i^{th} contaminant, $[g/m^3]$

Q_{vi} = The emission rate of the i^{th} contaminant, $[g/m^2\text{-}s]$

U = Average wind speed in the box, $[m/s]$

H = Height of the box, $[m]$

W = Crosswind width of the area source, $[m]$, and

A = Size of the area source, $[m^2]$.

An average wind speed value of 4.5 m/s speed was determined using the meteorological data from the nearby Newark International Airport in Newark, NJ. The dimensions A and W were determined using the square area source dimensions as described earlier. The input parameters used to determine the on-site concentrations were: H = 8.8 m; U = 4.5 m/s; and Z_0 = 0.1 m.

ISCLT AIR DISPERSION MODEL

EPA's Industrial Source Complex Long-Term (ISCLT) dispersion model was used to estimate off-site annual average concentrations of the compounds

released from the Carlstadt site. The ISCLT model is part of EPA's UNAMAP family of models which are considered to be EPA's preferred group of air models. It is a steady-state Gaussian plume model which can be used to assess pollutant concentrations from a wide variety of sources (USEPA 1986). ISCLT estimates annual average ground level concentrations in all directions around an emission source out to 50 km.

The first step in the process is to link the appropriate STAR meteorological data with the ISCLT model. Star data represent summaries of the observed joint frequency of occurrence of wind speed and direction for a range of atmospheric stabilities. Due to its close proximity to the Carlstadt site, STAR data from the Newark International Airport, Newark, NJ, compiled for 1955-1964 were used. Although this is an older meteorological record it should still be representative of the expected climatological conditions at the site.

ISCLT allows selection of atmospheric dispersion coefficients representative of a rural or more turbulent urban environment. Due to the location of the Carlstadt site, the urban setting was chosen for this assessment. ISCLT also accepts a user-specified receptor network. A polar receptor network was specified with 16 radials, one radial for every 22.5 degrees of azimuth. Receptors were located along each radial beginning at 100 m from the site and continuing out to a distance of 10 km. This resulted in a total of 576 receptors which encircled the emission source. All receptors were assumed to be located at the same elevation as the emission source. Because this assessment is limited to inhalation, no deposition rates were calculated. A source height of 0.0 m, representing a ground-level source, was input. The emissions from the Carlstadt site were assumed to be neutrally buoyant. The plume rise of emissions from the site were calculated using the final rise equations in the ISCLT model.

ISCLT treats area sources as squares. Since the area source at the Carlstadt site is not square, an effective area source was computed. The effective area source is found by taking the square root of the total area for the irregularly shaped Carlstadt site emission source. This value would then

be one side of the square effective area source. For the Carlstadt site the area of the emission source used in the ISCLT model was 168 m X 168 m. For the emission source, a unit emission rate was input (1 g/m^2/s). When the ambient concentration resulting from the unit emission rate is multiplied by the chemical specific emission rate in g/m^2/s, the result is the ambient concentration at that point for the specific chemical emission. For this risk assessment, maximum concentrations calculated by the ISCLT model along different azimuths were used to determine the exposure point concentrations for off-site workers and off-site residents. This was done because the site is located in an area where there are both industrial sites as well as residential areas. The maximum value for off-site workers occurred just outside the site boundary. The maximum value for the nearest off-site resident occurred approximately 1 kilometer northeast of the site. The maximum ambient concentrations from the model runs for the area source with a unit emission rate (1 g/m^2/sec) were predicted to be 28,153 ug/m^3 for an off-site resident and 592,352 ug/m^3 for an off-site worker.

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APPENDIX C

EXPOSURE AND RISK CHARACTERIZATION METHODOLOGY

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C. EXPOSURE AND RISK CHARACTERIZATION METHODOLOGY

The purpose of this appendix is to present the methodology used to estimate exposures and risks in the text of the Endangerment Assessment for the SCP site.

C.1 DIRECT CONTACT WITH CONTAMINATED SOILS

Chronic daily intake (CDI) estimates for incidental soil ingestion are calculated as follows:

$$CDI = \frac{(C_s)(I)(AI)(F)(YR)(X)}{(BW)(DY)(YL)}$$

where

CDI = chronic daily intake (mg/kg/day);

C_s = chemical concentration in soil (mg/kg);

I = amount of soil ingested (mg/day);

AI = relative oral absorption factor;

F = frequency of exposure (days/yr);

YR = years of exposure (years);

X = conversion factor $(kg/10^6 \text{ mg})$;

BW = average body weight (kg);

DY = days in a year (365 days/year); and

YL = years in lifetime or in the period over which risk is being estimated (70 year lifetime for carcinogens, period of exposure for noncarcinogens).

CDIs for dermal absorption of chemicals of potential concern are calculated as follows:

$$CDI = \frac{(C_s)(CD)(F)(YR)(Z)(ABS)}{(BW)(DY)(YL)}$$

where

CDI = chronic daily intake (mg/kg/day);

C_s = chemical concentration in soil (mg/kg);

CD = contact rate for soil (mg/day);

F = frequency of exposure (days/year);

YR = years of exposure;

Z = conversion factor (kg/10^6 mg);

ABS = dermal absorption factor;

BW = average body weight (kg);

DY = days in year (365 days/year); and

YL = years in lifetime or in the period over which risk is being estimated (70 year lifetime for carcinogens, period of exposure for noncarcinogens).

The total CDI associated with direct contact with soils is the sum of the CDIs from incidental ingestion and dermal absorption.

C.2 EXPOSURE TO CONTAMINANTS RELEASED FROM SOILS VIA INHALATION

The equation used to estimate the CDI through inhalation is:

$$CDI = \frac{(C_a)(V)(F)(YR)}{(BW)(DY)(LT)}$$

where

CDI = chronic daily intake (mg/kg/day);

C_a = ambient air concentration (mg/m^3);

V = ventilation rate (m^3/day);

F = frequency of exposure (days/yr);

YR = years of exposure (yr);

BW = body weight (kg);

DY = days in a year (365 days/yr); and

YL = years in lifetime or in the period over which risk is being assessed (70 year lifetime for carcinogens, less than lifetime for noncarcinogens).

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C.3 INGESTION OF GROUNDWATER

The CDI estimates for ingestion of groundwater were calculated as follows:

$$CDI = \frac{(C_w)(I)(F)(YR)}{(BW)(YL)}$$

where

CDI = chronic daily intake (mg/kg/day);

C_w = chemical concentration in groundwater (mg/liter);

I = amount of water ingested (liter/day);

F = frequency of exposure (days/yr);

YR = years of exposure (year);

BW = average body weight (kg); and

YL = years in a lifetime (70 years).

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